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Original Articles and Clinical Cases.

ON THE ENDOGENOUS OR INTRINSIC FIBRES IN THE LUMBO-SACRAL REGION OF THE CORD.

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For a number of years after it was ascertained that the posterior columns of the spinal cord were composed of two great tracts—an internal (column of Goll or postero-median column) and an external (column of Burdach or postero-external column)—it was generally supposed that these two tracts were found in the lumbar and sacral regions as well as in the cervical and dorsal portions of the cord, in which they were first described. Some text-books of anatomy and nervous diseases still make this statement, or if they do not do so directly, at least leave this opinion to be inferred. It has gradually, however, come to be recognised that this view is erroneous, and that in the lumbar and sacral regions it is not possible to separate a distinct postero-median column from a postero-external column in the same manner as can be done in the dorsal and cervical regions in cases of ascending degeneration. The differentiation of the two tracts does not become completed until the level of the eleventh dorsal segment is reached. Below this level the long, intermediate, and short ascending fibres (of Singer and

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Münzer) are intermingled, since the long fibres which pass from the lower posterior root ganglia to the medulla have not yet all reached the position near the posterior median septum which they ultimately occupy when they enter the postero-median column. In the second place a considerable proportion of the fibres of the column are not continuous with the posterior roots, but are derived from nerve cells in the grey matter of the cord itself. These fibres have been termed by Marie *endogenous* fibres, and it is to these that this article is devoted.

These *endogenous* fibres form two very well-marked tracts, one lying in the anterior part of the posterior column in close apposition to the posterior cornu, commissure and septum; the second in immediate relationship to the posterior median septum, and in part, to the surface of the cord. The former of these has been termed by Marie (1) the *cornu-commissural tract*, to indicate that it was regarded as connecting different levels of the grey matter of the cord. The German title dorsales Hinterstrangsfeld, indicates its topographical relations. The second tract has been termed by Dr. Muir and myself (19) the *septo-marginal tract*, the name being designed to indicate its relationship to the posterior median septum, in the same manner as the sulco-marginal tract of Marie is related to the anterior median sulcus. Obersteiner has also termed this tract the dorso-median sacral bundle (dorso-medianes Sacral-Bündel) and Edinger has called it the medianes Hinterstrangsfeld.

In order that a set of fibres may be regarded as of endogenous origin, there must firstly be an absence of degeneration in it when the posterior roots (or exogenous fibres) are divided or destroyed; and (secondly) it should itself degenerate when that portion of the grey matter from which it arises is atrophied or necrosed. These two conditions hold good in the case of both of the tracts mentioned.

The *cornu-commissural tract* is spared in locomotor ataxia, [which is now by very many neurologists recognised as a disease of the posterior roots,] even in its advanced stages, and in injury to, or compression of the cauda equina. On the other hand, it is found to undergo degeneration in

conditions which lead to the atrophy, or degeneration of the cells in the posterior cornu. Thus Ehrlich and Brieger, and Singer and Münzer (4) after causing necrosis of the nerve cells of the lumbo-sacral region by compression of the abdominal aorta, and thus cutting off their blood supply, found a distinct but somewhat diffuse degeneration throughout the whole of the posterior columns, but most marked in the anterior part of these columns. In pellagra, which is regarded as a disease of the grey matter of the cord, Tuczek (10) and Marie (1) found it distinctly affected. Still more recently Schlesinger (11) has shown that it is frequently degenerated in syringo-myelia, a disease which tends to destroy the grey matter, especially that portion of it in proximity to the central canal. Lastly, Fajersztajn (9) has shown that it is composed of fibres of short course, the majority of which have a descending direction. On the other hand, Déjérine and Spiller (7) maintain that it contains a considerable number of ascending fibres derived from the posterior roots.

Sections derived from advanced cases of locomotor ataxia are particularly well suited for the study of this tract. When these are stained by the Weigert-Pal method it stands out with great distinctness against the degenerated fibres. It extends through the whole of the lumbo-sacral region of the cord, being traceable from the lowest dorsal segment to the extreme tip of the conus medullaris. It attains its greatest size at the level of the lower lumbar region, and diminishes somewhat rapidly above and more gradually below this level. Throughout its whole longitudinal extent it stands in close relation to the posterior commissure, and (except perhaps in the lowest sacral segment) to the anterior part of the inner margin of the posterior cornu. Its inner margin, below the third lumbar segment, lies in contact with the posterior median septum. Above this it becomes gradually displaced outwards by the ascending fibres of the posterior roots of the lower spinal nerves, as they pass upwards to enter the postero-median column. When this latter column is fully developed, *e.g.*, at the level of the eleventh dorsal segment, what remains of the cornu-commissural tract is displaced

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from the septum completely, and also from that portion of the posterior commissure which lies immediately in front of the tip of the postero-median column. Posteriorly the tract has no definite margin. Its outer portion merges gradually into the part of the column behind it (the so-called middle root-zone of Flechsig), while in the neighbourhood of the septum, below the level of the first sacral segment, its inner portion becomes fused with the septo-marginal tract, in such a way that in sections derived from ataxia it cannot be determined where the one ends and the other begins.

A fuller idea of the topographical relations of the tract may be obtained by a study of figs. 1—9, which have been made from careful drawings of Weigert-Pal sections of an advanced case of ataxia. It will be seen from these that at the fifth sacral segment (fig. 9), the tract is in contact with the posterior commissure and the anterior part of the septum, but that it does not quite reach the cornu, being separated from it by a narrow area of degenerated fibres. Posteriorly it passes, without any line of demarcation (fig. 9), into the septo-marginal tract, appearing to form with it a continuous tract extending from the commissure to the periphery.

At the fourth sacral segment (fig. 8), the external margin has reached the cornu and extends along it backwards as far as the gelatinous substance of Rolando. The other relations are unaltered. The tract is not yet of any great extent, and the apex of the column is narrow and pointed.

In the upper sacral segments (figs. 7, 6, 5), the tract has considerably increased in size. The external margin extends further backwards along the posterior cornu, and the inner border passes along the septum to become fused with the septo-marginal tract.

At the fourth and fifth lumbar segments (figs. 4 and 3), at which levels, as already stated, the tract seems to have attained its maximum size, the inner margin does not extend so far along the septum, and is now distinctly differentiated from the septo-marginal tract. This latter is now limited to a small area at the posterior half of the septum. The separation of the two tracts seems to be effected

by the ascending (in this case degenerated) fibres of the posterior roots, which are becoming applied to the septum in their passage upwards to aid in the formation of the column of Goll. As additional fibres from the posterior roots become applied to these at higher levels (upper lumbar, fig. 8), the separation of the cornu-commissural and the septo-marginal tracts gradually increases (as in ataxia these root-fibres are degenerated the intervening space is paler than either of the tracts in question). The cornu-commissural tract is displaced outward, till at the twelfth and eleventh dorsal segments (fig. 1), it comes to form a narrow band bordering the posterior commissure and grey matter of the posterior cornu.

Above this level it is not easy to distinguish it as a distinct tract, but it apparently diminishes rapidly in size. The fibres which compose it can be seen entering it from the grey matter of the posterior cornu, and almost exclusively from that of the same side.

The *septo-marginal descending tract* also remains undegenerated in ataxia, and its position very closely corresponds to the area of fibres which Dr. Muir and I (19) found to degenerate after a crush of the cord at the level of the upper lumbar segments. In this case very little degeneration was found in the cornu-commissural tract, a proof that the greater part of the fibres of the latter arise below this level, while many of the septo-marginal tracts have a higher origin. This is in agreement so far with Hoche's observations as to the high origin of the tract. [The lesions in Hoche's cases were respectively below the level of the eighth cervical, and between the fourth and fifth dorsal segments (12).] It also enables us to indicate where the septo-marginal and cornu-commissural tracts meet each other. The fact that it has degenerated in a downward direction, renders it quite certain that it is not an upward continuation of posterior root fibres, and this has been corroborated by the observation of Pineles (23), that it remains unaffected in ataxia affecting the sacral segments. It stands out from the degenerated fibres in the lumbo-sacral portion of the cord even more distinctly than does the cornu-commissural tract.

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At the level of the lowest sacral root (fig. 9), the undegenerated area in question is situated at the postero-internal angle of the posterior column. It forms a racquet-shaped area, or a triangle with the edges rounded off, two of its sides being applied to the posterior median septum, and to the periphery respectively, and the third forming a somewhat indefinite margin between the degenerated and undegenerated parts of the cord, which at this level are not sharply differentiated from each other. The anterior angle of the tract seems to merge into the undegenerated area lying in the anterior part of the column—the cornu-commissural tract. The remainder of the posterior columns is not so degenerated as at higher levels, so that the healthy areas do not stand out so clearly. In the next segment, the fourth sacral (fig. 8), the tract has greatly altered. It has become narrower, and much elongated antero-posteriorly. In the greater part of its extent it forms a very narrow band along the posterior median septum, which as it nears the periphery, suddenly widens out into a club-shaped head. This head is rendered more prominent by the apparent shrinkage of the degenerated areas of the column, which produces a retraction of this part of the periphery of the cord from which the rounded club-like head of the tract projects boldly, the depression thus formed in the outline of the cord being filled in by the connective tissue and vessels of the thickened membranes. It is seen in the illustration as a pale area on the outer side of the projecting head. At this level the undegenerated area is very distinctly marked off from the remainder of the column. In the third sacral segment (fig. 7), there is very little change in the appearance of the tract, what change there is being in the shape of the head. This instead of being rounded is almost pointed, like the head of a broad spear, and projects further beyond the remainder of the column than in the former section. The recession of the periphery is deeper and more angular, and is as before, filled up by the thickened membranes. In this, as in the previous sections, the anterior end of the tract becomes merged in the fibres of the undegenerated cornu-commissural tract.

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At the level of the upper sacral segments (figs. 6 and 5), the tract has become distinctly larger, the band along the posterior median fissure being broader and the head also larger. The head still retains its pointed appearance, but does not project beyond the general line of the periphery of the cord: the angular depression at its outer side is still filled up by connective tissue. The tract continues to be sharply differentiated from the remainder of the column and to merge in the cornu-commissural tract anteriorly.

When the fifth lumbar segment is reached (fig. 4) there is again a great change in the appearance of the tract. It has greatly diminished in extent, now occupying only about a third of the length of the posterior median fissure instead of the greater part of the length; and, further, it no longer reaches either the cornu-commissural tract or the periphery of the cord, but forms a narrow tract, with a rounded posterior extremity and tapering to a point anteriorly. Roughly speaking, the shape resembles that of an Indian club. The broad posterior end bulges somewhat toward the posterior median septum, beyond the general surface, and a small interval is thus formed behind it, between the two posterior columns; this is filled up by connective tissue as in the previous sections. At this level the tract corresponds fairly closely to the "oval field" of Flechsig.

In the fourth lumbar segment (fig. 3) the tract is further diminished in size, and altered in position. It has passed backwards again, and forms an angular band outlining the postero-internal angle of the cord, one limb being placed along the periphery, the other along the posterior median septum for rather less than a fifth part of its extent. The tract is thus separated by a considerable interval from the cornu-commissural tract, which is well marked at this level.

In the segment above this, the third lumbar (fig. 2), the tract is still diminishing. It is no longer applied to the posterior median septum in any of its extent, but forms a very small, narrow band, of which one extremity touches the middle line, the other terminates gradually a short distance along the periphery. The tract has now become

scattered, with no very definite line of separation from the degenerated areas.

The final position taken up by the tract is seen at the level of the twelfth dorsal segment (fig. 1). Here the tract is represented by a small group of fibres, somewhat triangular in shape, placed at the periphery of the cord, about one-third of the distance from the middle line to the posterior roots. The fibres are few in number, but constitute a perfectly well defined tract seen in many successive sections. Lying anterior and external to it is an area containing more diffusely scattered healthy fibres.

The tract cannot be traced clearly higher than this level in cases of locomotor ataxia, but Hoche's case of degeneration after a lesion in the lowest cervical segment (14) has proved that it may originate as high as this. Above the eleventh dorsal segment, however, it appears to become diffused within the hinder part of the postero-external column, and is therefore not so easily followed as at lower levels. The evidence as regards its origin is thus still insufficient, and it can only be said at present that the tract originates in part in the higher segments of the cord, gains greatly in bulk about the level of the first sacral segment, and terminates in the lower sacral and the coccygeal region, by sending fibres forward along the posterior median septum into the grey matter near the central canal where they are lost amongst the other fibres in this part. The endogenous nature of many of its fibres is proved by the fact that in the case of ataxia described by the writer, there was a great accession to the number of its fibres at the first sacral segment, in spite of the almost complete degeneration of the posterior roots for some distance above and below this level.

It is very probable that at one part of its course, *i.e.*, in the lower lumbar segments, the tract corresponds to the "oval field" described by Flechsig, since at this level it occupies a small area close beside the posterior median septum and at some distance from the periphery, which when taken together with its fellow of the opposite side is oval in form. The discrepancies in the descriptions given

by the various authors as to the exact level at which this oval field appears are no doubt due in part to differences in the situation and nature of the lesion and in part to the different methods of staining employed. This also applies to some other slight differences in the descriptions.

The tract is undoubtedly mainly a descending one, but Déjérine and Spiller have concluded that it also contains some fibres which degenerate upwards after injury to the cauda equina. This is very probable, but there are, as yet, so few cases on record in which this tract has been accurately described, that on this and on many other points it is quite impossible as yet to form a definite conclusion.

As regards the cells from which the cornu-commissural and the septo-marginal tract are derived, it is not possible as yet to make a definite statement.

Cajal first showed that in the chick some cells of the posterior horn sent their axis-cylinder processes to the posterior columns, and Lenhossek has been able to demonstrate similar cells in the guinea-pig, etc., as well as in the human embryo. They are situated at the base of the horn and in the Rolandic substance. None of the cells of Clarke's column send their processes to the posterior column.

With regard to the function of these two tracts nothing definite can be stated further than that they are longitudinal and commissural in character. Their position strongly points to their being connected with the lower organic reflexes.

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DESCRIPTION OF PLATES.

The lettering is the same throughout.

c.c. = Cornu-commissural tract.
s.m. = Septo-marginal tract.

The figures are reproduced from drawings of the posterior columns made by Dr. Jessie M. MacGregor.

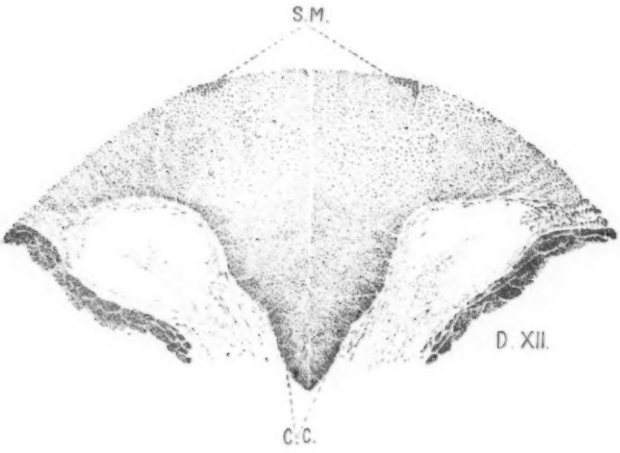

FIG. 1.

Level of twelfth dorsal segment.

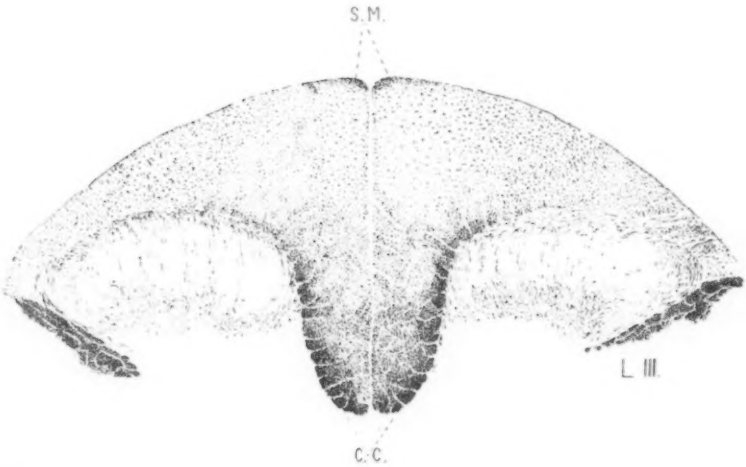

FIG. 2.

Level of third lumbar segment.

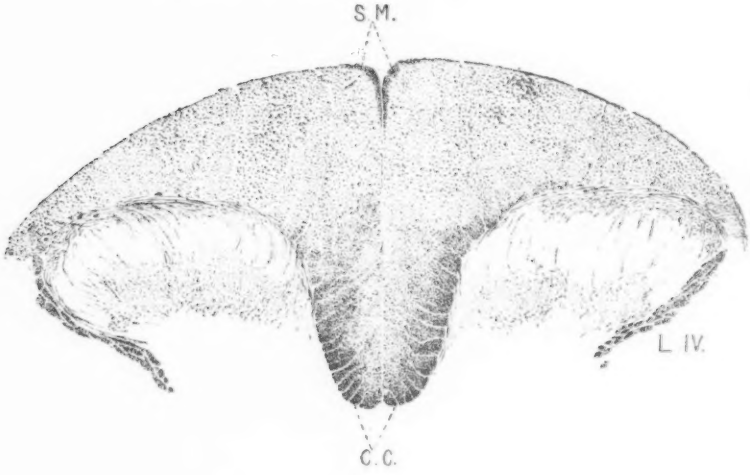

FIG. 3.
Level of fourth lumbar segment.

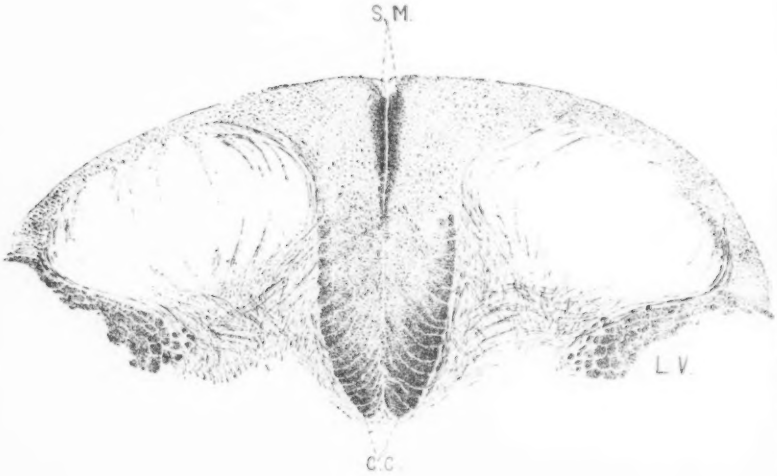

FIG. 4.
Level of fifth lumbar segment.

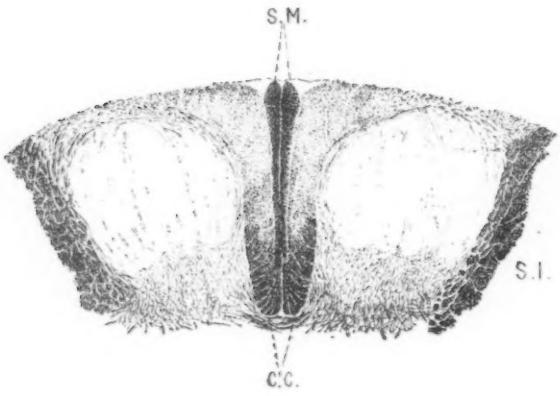

FIG. 5.
Level of first sacral segment.

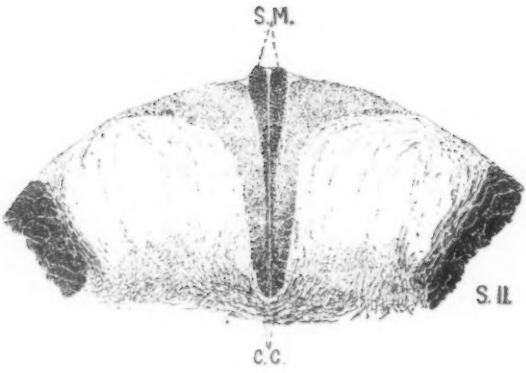

FIG. 6.
Level of second sacral segment.

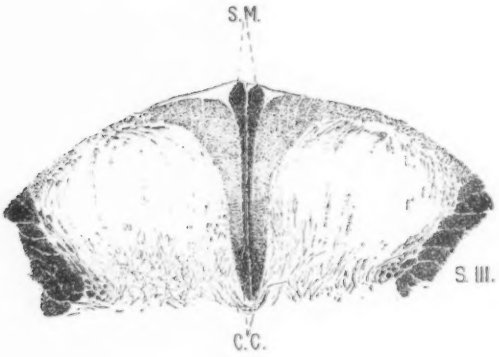

FIG. 7.

Level of third sacral segment.

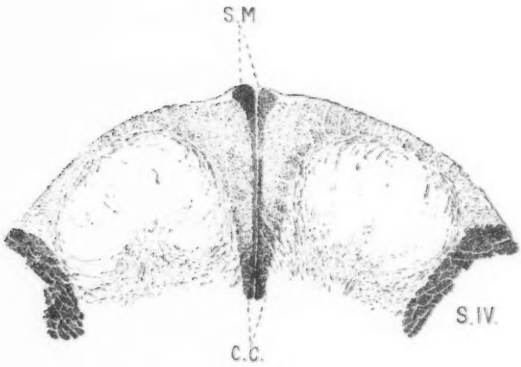

FIG. 8.

Level of fourth sacral segment.

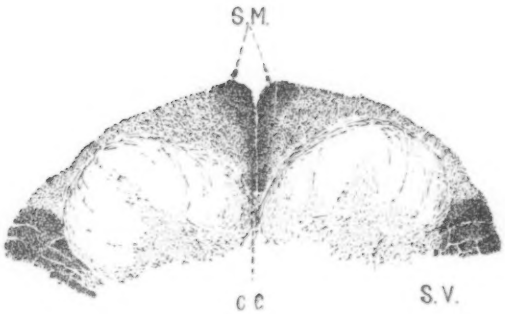

FIG. 9.

Level of fifth sacral segment.

THE MORBID ANATOMY OF A CASE OF HEREDITARY ATAXY.

(No. VI. of Dr. Sanger Brown's Series of Cases).

BY DR. ADOLF MEYER, WORCESTER, MASS.

WITH AN INTRODUCTION BY DR. SANGER BROWN.

By way of introduction to Dr. Adolf Meyer's Anatomical Report, and with the purpose of rendering the same more easily understood by those who have not read, or who do not distinctly remember, my clinical report of the series published in 1892, in BRAIN, and in the *North American Practitioner*, I now reproduce my original diagram, showing how the disease was transmitted, and the ages at which the respective subjects of it were attacked, with other data calculated to give a fairly good general idea of the nature of the symptoms, and a more particular clinical report of the case which furnished the material for Dr. Meyer's report.

For the purpose of giving a general idea of the symptoms in my series, perhaps I cannot do better than to quote from my original paper:—

"Taking these cases alone for a text, and assuming them to be cases of hereditary ataxy, the following diagnostic criteria might be fairly deduced:—

"Hereditary ataxy is a disease which may be traced through several—at least four—generations, increasing in extent and intensity as it descends, tending to occur earlier in life, and advance more rapidly. It usually attacks several members of the same family. It occurs most frequently between the ages of 16 and 35, but it may begin as early as 11 and as late as 45. It shows no marked preference for sex, but it descends through females four times as frequently

as through males. The influence of an exciting cause can rarely be demonstrated, but in some instances a fall or injury has appeared to determine the onset; and any cause like child-bearing or lactation, which very much depresses the vital forces, may produce a rapid advance of all the symptoms. There is always considerable incoördination of all the voluntary muscles, and a sluggishness of the movemeets, which they produce when the disease is well established. This is usually noticed first in the muscles of the legs, but in a few months or years extends to the arms, face, eyes, head, and organs of speech. Sometimes it occurs first in the upper extremities, and sometimes in the organs of speech.

"The ataxy is often extreme, and the gait devious, the patient deviating several feet on either side of the intended line of progression, before he loses the power of walking. The ataxy is not markedly increased by closure of the eyes. The sense of posture is perfect.

"Some weakness of the muscles of the legs, without atrophy, is frequently an advanced symptom, and occasionally there is permanent spastic contraction of the legs. In developed cases there are usually choreiform movements of the head, and often of the arms, accompanying all voluntary movements. These irregular movements occur in the hands, legs, or head whenever it is attempted to maintain either of these parts in a fixed position by a voluntary muscular effort.

"There is usually some degree of static ptosis, with overaction of the levator on looking upward. In rare cases there may be temporary diplopia in the early stages, due to weakness of the external rectus. There is no nystagmus of any kind.

"Atrophy of the optic nerve is a constant and early symptom, and usually progresses slowly with the other symptoms. Rarely it begins earlier in one eye than the other.

"The response of the iris to light and accommodation is sluggish, and diminishes with the advance of optic nerve atrophy. When this latter is complete, as may happen in

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advanced cases, there may be complete internal and external ophthalmoplegia.

"There is always marked disturbance of the articulation, probably due to incoördination of the muscles concerned, for weakness cannot be demonstrated. In some cases there is a troublesome tendency to strangulation in swallowing liquids, due to their getting into the larynx, but otherwise swallowing is in no way difficult.

"Occasionally the sphincters are slightly, but positively, affected, this symptom only appearing in those cases where spontaneous pains in the legs co-existed, having some of the characteristics of those occurring in locomotor ataxy. Excepting the spontaneous pains already mentioned, there is no disturbance of sensibility. There are no vaso-motor or trophic symptoms, but there is a marked tendency to emaciation; there is no hypertrophy or valvular lesion of the heart.

"The knee-jerk is always exaggerated, and there is frequently ankle-clonus, and the cutaneous reflexes are also always exaggerated, but to a less degree. The exaggeration of the reflexes is an early symptom, and they often decline considerably when the disease is far advanced.

"There is never paralytic club-foot, nor any other deformity, excepting rarely permanent spastic contractions of the legs in advanced cases. In none of these cases have the patients ever suffered from rheumatism, so far as I can learn.

"I wish to repeat that the above summary of symptomatology of hereditary ataxy is only intended to apply to this particular series of cases; and I have only presented it in this way so that it might be the more easily compared with other series."

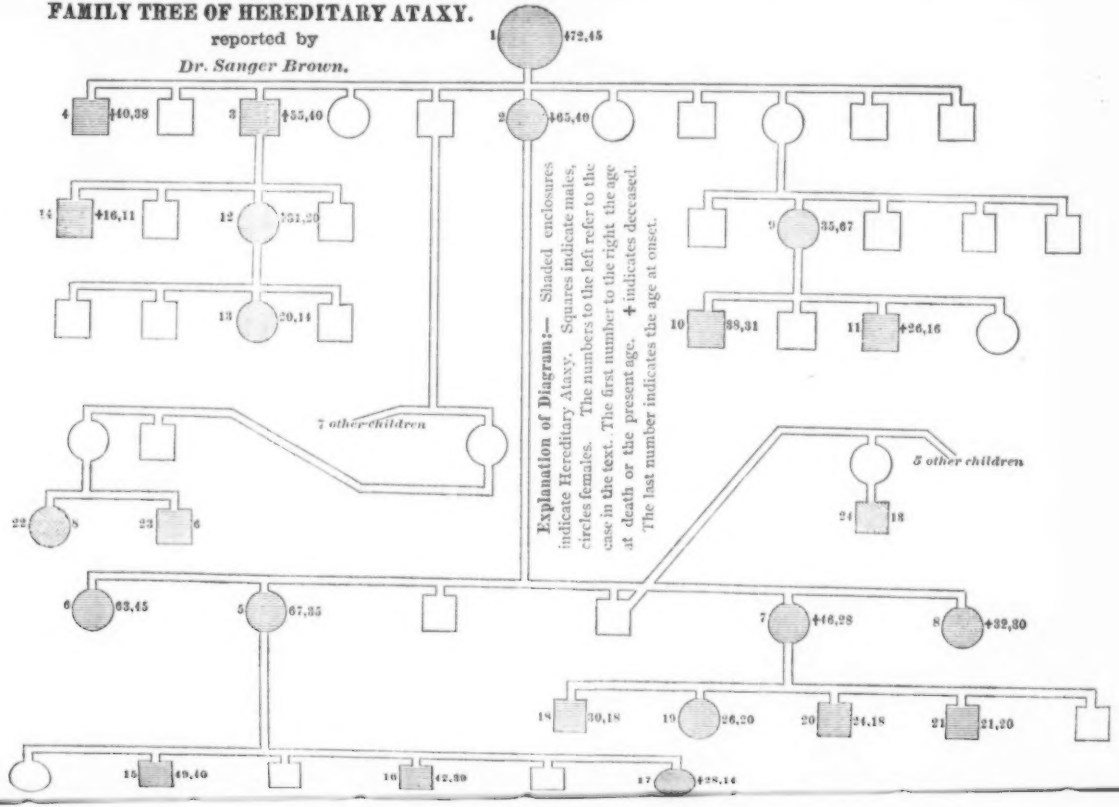
I shall again quote from my original paper the description contained of case vi., upon which Dr. Meyer's report is based.

"*Case VI.*—Female, age 63. First symptoms at 45, consisting of ataxy of legs. The course of the disease has been slow. She can still walk on an even surface without assistance, but has to go very slowly and sways about considerably, usually steady-

FAMILY TREE OF HEREDITARY ATAXY.

reported by

Dr. Sanger Brown.



Explanation of Diagram:— Shaded enclosures indicate Hereditary Ataxy. Squares indicate males, circles females. The numbers to the left refer to the case in the text. The first number to the right the age at death or the present age. + indicates deceased. The last number indicates the age at onset.

ing herself by holding on to furniture. The hands are certainly involved, but not greatly, as she can still do needlework and pour tea. Vision is considerably affected, but she can read coarse print in dim light. Cannot read in a bright light. Speech has become very slow, but is quite distinct. General health fairly good. Other symptoms negative. History given by a relative, himself affected, who has seen her almost daily for years."

This person died at the age of 67, of tubercular diarrhœa, from which she suffered for several months. About four months prior to her death, the neck of the femur was fractured by a fall, after which she was unable to leave her bed. Dr. R. L. James had been her medical attendant for over a year prior to her death, and noted very carefully the symptoms pertaining to her nervous disease, which he was particularly well qualified to do, inasmuch as he had in the past four years been medical attendant to, and seen almost daily, socially or professionally, two nephews and a niece of the patient who suffered from the same disease. He states that the knee-jerks were greatly exaggerated, but that there were no sensory symptoms of any kind; that there had been marked failure of vision two years at least before death, so that the patient was unable to read, but could see sufficiently well to play cards. She had lost the power of walking alone for at least two years before breaking her thigh, and for the last year she had been unable to do needlework or pour tea, though she could still manipulate cards fairly well. The facial expression for the last eight or ten years of life was highly characteristic, that is to say, the partial ptosis and relaxation of the facial muscles, being suggestive of somnolence when in repose, but when in animated conversation there was over-action of some of the muscles of expression and under-action of others, giving the face a very peculiar appearance. There was never any evidence of mental disorder.

The autopsy was performed under great difficulties, and the brain reached me in a rather lacerated condition, in Müller's fluid. Unfortunately there are no data on hand on the autopsy, the fresh appearance of the tissues, the weight of the brain, and especially the cerebellum. The

latter was centrally softened, as far as could be ascertained, owing to the slow action of Müller's fluid in the very warm weather. The spinal cord was well preserved. Of the brain, the medulla oblongata, cerebellar cortex, and the optic nerves and tracts were taken for examination. In absence of macroscopic description we proceed at once to the study of the sections.

Spinal Cord.—Sections from the lumbar, lower and upper thoracic and cervical cord were made. For the study of the corpora amylacea a short fixation in Müller's fluid, and subsequent alcohol hardening proved satisfactory for a stain with Bohmer's hæmatoxylin. The other specimens were completely hardened in Müller's fluid, and, with or without a total stain in a 1 per cent. carminate of sodium solution, dehydrated and embedded in the usual manner. The stain for the medullated fibres, was Wolter's modification of Weigert's method, with or without counter-stain by 0·5 per cent. acid fuchsine.

The cross-section of the cord is not thinner than might be expected in a woman of 67. The diameters are as follows:—

	Trans-verse.	Antero-posterior.
Cervical enlargement ...	10·8 mm.	8·5 mm.
Mid-dorsal region	7·0	7·0
Lumbar enlargement ...	9·5	8·0

The specimens stained with Böhmer's hæmatoxylin after a short fixation in Müller's fluid, show a remarkable number of corpora amylacea throughout the spinal cord, the bodies are distributed very much as Redlich represents them in his drawing.[1]

They are most numerous in the dorsal half of the cord, especially in the entrance zone of the posterior roots, where they are arranged in strings of several layers; further along the posterior septum, in the subpial neuroglia layer, along blood vessel septa, and all through the posterior columns. In the lateral columns they keep near the surface; they are

[1] E. Redlich, Die Amyloidkörperchen des Nervensystems. *Jahrbücher f. Psychiatrie.*, vol. x., 1892, p. 23.

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relatively rare in the anterior columns and anterior horns and practically absent in the rest of the gray matter and in the ground bundles. In one posterior root zone over two hundred can be counted readily. They vary in size, and are easily distinguished from the much smaller nuclei. As Redlich, Koller and others have shown, they occur frequently in old people, but I have not been able to obtain such an excessive number in any of my cords, nor could any one show me similar specimens. We must say though that in the ordinary staining methods the granules do not present themselves as clearly as in a specimen which was not very long in Muller's fluid. The material of comparison is therefore not quite fair.

The neuroglia and the blood vessels were examined with a stain of Ehrlich's haematoxylin and subsequent counter-stain with picric-acid fuchsine solution. This stain gives a very satisfactory bright colour to the mesoblastic connective tissue, while the neuroglia proper remains a duller red.

The pia is somewhat thickened, from 0·1—0·15 mm. over the lateral columns, but only about 0·025—0·04 over the dorsal columns, and 0·04—0·05 over the anterior. The blood vessels are normal in number, and there is very little if any hyaline degeneration of the muscularis of the larger ones.

The neuroglia is somewhat increased in the whole extent of the subpial neuroglia layer. The latter measures about 50μ over the dorsal columns, about 25μ over the ventral half, and over the dorsal part of the lateral columns and the posterior root zone it varies between 40 and 100μ. Fromann[1] says the diameter of the neuroglia margin varies between 0·01 and 0·06 mm., according to Goll from 0·02 to 0·03 mm. Kölliker[2] states that the layer varies from 0·022 to 0·045 mm. There is no doubt, therefore, that the increase is fairly well marked in our case. In the region of the pyramidal decussation this is quite plain, since the thickening is limited to the extent of the cerebellar tract.

[1] Fromann, Untersuchungen über die normale und pathologische Anatomie des Rückenmarkes. I. Theil, 1864, p. 28, quoted by Weigert, Beiträge zur Kenntniss der normalen menschlichen Neuroglia, Frankfurt, 1895, p. 146.

[2] Kölliker, Handbuch der Gewebelehre des Menschen, 6. Aufl. Vol. II. 1896, p. 151.

The neuroglia of the rest of the cord will be spoken of in the analysis of the various tracts.

Lumbar Cord.—The roots are quite normal. *The posterior columns* have a normal proportion of thick and thin fibres. It is hardly possible to detect anything abnormal; perhaps the neuroglia septa are slightly broad in the central portions of the posterior columns. But it is impossible to make out anything but a faint similarity with Flechsig's[1] or Marinesco's[2] figures since the roots, and especially the lateral part of the middle root zone are quite normal; only the mesial part of Flechsig's middle root zone may have a slight increase of neuroglia.

The amount of fibres and collaterals is quite normal in the gray matter; the motor cells, though pigmented, show mostly a well defined nucleus near the centre of the cell body.

The antero-lateral columns are densest close to the gray matter (ground bundles). The region of the pyramidal tracts shows very few thick fibres, but no striking increase of neuroglia.

In the *Thoracic* region nothing deserves special notice besides the condition of Clark's columns. The number of their cells is decidedly small. The cells that remain are partly normal, partly show a change which I noticed often in general paralysis and elsewhere, and which Marinesco[3] has lately described in cases of locomotor ataxia.

The normal cells, very few in number, have the nucleus round, and in the centre or near it. The granulations, of course, are not visible, since the hardening with Müller's fluid rarely preserves them. The larger number of the cells are more vesicular; the nucleus is pressed to the periphery and is crescent shaped. There are further a few cells which are quite shrunken and have no differentiation. There can

[1] Flechsig, Ist die Tabes dorsalis eine "System-Erkrankung." *Neurolog. Cbl.* Vol. IX. 1890, p. 73, fig. 6.

[2] Marinesco, "Lésions de cordons postericurs d'origine exogéne." Part V. of "Babes' Atlas," plate VII.

[3] Sur une particularité de structure des cellules de la colonne de Clarke, et sur l'état de ces cellules dans le tabès simple et dans le tabès associaé à l paralysie générale, par G. Marinesco. *Revue Neurologique*, 4e Année, Nov. 15, 1896, p. 633.

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be no doubt that these findings stand in some connection with the condition of the cerebellar tract mentioned below.

The posterior and antero-lateral columns show no deep lesion. Their condition stands about midway between that found in the cervical and the lumbar regions with regard to the increase of neuroglia and the proportion of thick to thin fibres.

The Cervical Cord.—Here we find a number of very pronounced alterations.

The *posterior columns* show a field of degeneration along the middle of the septum; the superficial part of Goll's column is also affected, though less markedly, and, on either side, the transition area between Burdach's and Goll's tracts

is somewhat rarefied. In the normal cord most of the fibres in these regions are of moderate thickness, forming plain rings with the Weigert stain; thin fibres, with almost no lumen, are rare. In our sections many of the thick fibres are gone, and also the thin fibres are scarcer. The neuroglia is correspondingly increased, the blood vessels are a little wider and surrounded by thick neuroglia masses. The changes are somewhat more diffuse and less marked than in tabes.

The *lateral columns* of the *normal* cord show a layer of thick fibres plainly limited all along the surface of the pyramidal tract, but less distinctly outlined in the region of Gowers' antero-lateral tract. The distinction is formed by the fact that the fibres of the direct cerebellar tract have very few collaterals, while these are abundant in the pyramidal area and also in the ground-bundles and a little less in the antero-lateral columns. The collaterals show with the Weigert stain under a low power (Reichert or Leitz 3) as points without lumen; under a higher power (Reichert 7) they show a small lumen, except the smaller ones, which are point shaped.

In our case, we notice at once the diminution in size of the direct cerebellar tracts. Only relatively few large fibres are left, and they are separated by neuroglia. In the normal cord, this area, so free from neuroglia and collaterals, is about 0·4—0·5 mm. broad; in our sections, the atrophic area measures 0·15 mm. at the most on the side where the proliferation of neuroglia is less marked; on the other side it is of normal breadth (0·4—0·5 mm.), but containing much interstitial neuroglia, fewer fibres, and broader blood vessels. Farther ventrally beyond about the middle of the lateral columns the area loses its demarcation rather suddenly, and all we can see is a general deficiency of thick fibres along the surface. On the one side the bundle is rarefied *en bloc* for 2·2 mm. from the posterior root and superficially only with a little less demarcation for further 2·4 mm. (fig. 1).

The fibres which remain are very large, averaging a diameter of 10·15. Throughout the rest of the anterior and lateral columns there are only few fibres of this size in our

specimens, while they are fairly numerous in the normal throughout the pyramidal tracts and the antero-lateral tract area. The direct pyramidal tracts show a slight increase of neuroglia, the lateral pyramids lesser. It is hardly possible to make out Gowers' tract; all we can say is that the ground-bundles are denser and richer in collaterals than the more superficial layers.

The gray matter shows no evident alteration.

Transition into the Medulla.—All we have to note is the extension of the changes described; hardly any change in the pyramids, slight degeneration in the posterior columns, and especially degeneration of the direct cerebellar tract on both sides, not complete, but leaving a triangular area with scanty large fibres, broad neuroglia septa, and especially a well-limited thickening of the marginal neuroglia layer. The field is decidedly smaller than normal (it is easy to see it in the normal carmine specimens and also in Weigert sections). The thick fibres of the antero-lateral tract (ventral cerebellar) are also rare, but not kept apart by neuroglia.

Medulla Oblongata.—The medulla had been torn off from the pons at the autopsy and was not satisfactory for a careful examination of the topography. All we can say from the sections is that the area of the direct cerebellar tract continues as described and then disappears in the restiform body, where at best a slight rarefaction of fibres can be made out. The ventral cerebellar tract has few large fibres and is somewhat pale. The olives have a normal amount of fibres and cells. The pyramids are slightly less dense than normal.

Cerebellum.—Only pieces of the cortex of the hemispheres are examined. The folia are hardly atrophic as a whole. Nothing can be made out but, perhaps, a slight reduction of then umber of Purkinje cells in a few parts. This also occurs in normal specimens, although perhaps less than here. Other folia have a very large number of perfect Purkinje cells.

The *optic nerves* are free from degeneration, and also from neuroglia-overgrowth.

In the monograph of Londe,[1] the autopsies of Fraser,

[1] Londe, "Maladies familiales du système nerveux. Hérédo-ataxie cérébelleuse." Paris, 1895.—*See* BRAIN, 1895, p. 421.

Nonne and Menzel are discussed. Our case seems to be the first addition since Marie established the clinical picture of cerebellar heredo-ataxia. On account of the defective condition in which the brain reached the pathologist, several points cannot be as clearly decided as would be desirable; but the following statements are well founded.

1. There is no circumscribed cerebellar lesion, nor does the cortex show a marked decrease of the number of the Purkinje cells.

2. Parts of the spinal cord and medulla which are known to have relations with the cerebellum were found affected.

3. The spinal cord as a whole shows increase of the superficial neuroglia and a remarkably large number of corpora amylacea, similar to what is seen in very old people, in paralysis agitans, &c.

It would not be fair to build on this one observation a dogmatic theory of the disease of the family. We may, however, safely say this:

The separation of a *type cérébellaire* from the general picture of hereditary ataxia is clinically justified, but anatomically to less extent than Marie seemed to expect. In his case Nonne saw a simple hypoplasia of cerebellum and spinal cord and a peculiar preponderance of thin fibres in the anterior roots. Fraser found atrophy of the cerebellum, but no lesion of the cord (his case dates from a time when the Weigert hæmatoxylin stain did not exist). Menzel, however, had in his case even more varied lesions than we found: atrophy of the cerebellum with notable disappearance of Purkinje cells and changes of the dentate nucleus, changes in the spinal ganglia and posterior roots: degeneration of Goll's and Burdach's tracts, the pyramidal tracts and cells of Clarke's column; changes in the nuclei of the lateral and posterior columns, of several motor nuclei of the bulb, of the gray matter in the pons, the olives, corpus Luysii, and black substance of Sömmering.

The safest explanation is, that in our case we met with a diffuse deterioration of the afferent cerebellar system, with special involvement of the direct cerebellar tracts. Moreover, there is an affection of the peripheral sensory elements

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in their course within the neuraxis, but apparently only of those collaterals which connect the lower level with the higher (cerebral and cerebellar) apparatus. We could not otherwise understand the persistence of an exaggerated knee-jerk in our case, and the absence of degeneration of the dorsal columns in the lumbar region; the reflex collaterals are intact; only the cervical cord shows plainly a degeneration of Goll's column. In a similar way we have found in general paralytics a degeneration of the crossed pyramidal tracts of the lower segments of the cord, where the crus cerebi and the oblongata did not manifest a degeneration of the pyramid. Whether the great number of amyloid bodies should be interpreted as an excessively senile phenomenon cannot be decided, since the patient died rather old, and we have not enough control specimens; it is, however, fair to admit, that even for a woman of 67, the number of these bodies and the general increase of neuroglia was at least indicative of marked retrogressive changes. The cerebellum manifests a slight decrease of Purkinje cells. The optic nerves are intact. There is no marked degeneration of the pyramids, but a slight diminution of the thick fibres.

My general attitude in explaining diffuse nervous affections has been put forth in a paper on Landry's paralysis, published with Dr. Diller, in the *American Journal of Medical Sciences*, April, 1895; this view is already taken for heredo-ataxia both by Nonne[1] and by Edinger.[2] The latter says: "I am inclined to accept Londe's opinion, according to which one would do better to keep the two diseases together under the name of hereditary ataxia; the one form Friedreich's disease proper, should be diagnosticated where the disorder begins with spinal symptoms, while the name cerebellar heredo-ataxia should be reserved to the cases which start with cerebellar symptoms. Londe shows beautifully how Marie's type gradually associates itself with spinal symptoms, and Friedreich's with cerebellar ones. Moreover, cases are on record where both were attacked simultaneously. Schultze arrived apparently at similar conclusions. On the ground of the 'Ersatz

theorie,' I am of opinion that according to the varying localisation of the congenital weakness function will create various pictures."

That the character of the lesion is not identical in appearance with the typical gliosis of Friedreich's disease as described by Déjérine, must have its reasons in the difference in the age of onset, and the extent of the pathological process.

[1] Ueber einen in congenitaler beziehungsweise acquirirter Coordinationsstörung sich kennzeichnenden Symptomen-complex." *Arch. f. Pysch.*, vol. xxvii., p. 497.

[2] (Art. "Friedreichsche Krankheit." "Real-Encylop. d. ges. Heilkunde," 3 Aufl., 1895, vol. vii., p. 117).

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"SURVIVAL MOVEMENTS OF HUMAN INFANCY."

Being the substance of an address given before the Manchester Branch of the British Association for Child Study, March 2, 1897.

BY ALFRED A. MUMFORD, M.D.

WHEN we watch closely the limb-movements of human infancy and early childhood, we cannot but be struck with their peculiar purposeless character, and notice how widely they differ not only in form but even in rhythm from the voluntary movements of later date.

These early limb-movements as we see them immediately after birth, are presumably similar to those occurring in utero, though from the nature of the surroundings they necessarily present a greater range.

They are generally known as "Spontaneous" or "Involuntary" movements, sometimes as "Stretching movements." They steadily diminish in number and in importance till the end of the fourth or sixth month of independent life, by which time many have entirely disappeared, while others survive but in a modified form. In fact, on careful examination, a definite selective process is found to occur among them, by which some become utilized for special purposes, serving mainly for the expression of the various feelings and emotions. These are therefore permanently preserved. Others gradually diminish and tend to disappear, being replaced by the gradually evolving voluntary movements, which appear in successive stages or batches of increasing complexity throughout the early years of childhood. For the early spontaneous movements the writer suggests the term "Survival Movements," and besides describing their nature, proposes to attempt to give

some explanation of their origin. The full description of the definite stages in which the *voluntary* movements appear, and how they arise from and supplant these early survival movements must be left to another paper.

A careful study of these early infantile movements shows that though they are aimless as far as the individual infant performing them is concerned, they are not necessarily meaningless as regards the development of the race of which the individual infant is but an off-shoot. It is quite possible, indeed we might say probable, that they are vestiges of functions of the limbs which were of prime importance to the members of the race at another and early period of its growth, but began to lose their prime importance, and therefore their full development, when the forelimb gradually acquired other and higher functions. They remain, I believe, as true survival movements from earlier stages of existence, and thus serve to show the order in which the evolution of the human forelimb took place; just as many other bodily structures remain, such as the pineal gland and the vermiform appendix, which denote the existence of functions that are not now in activity.

Preyer,[1] who seems to have made the most thorough investigations into early infantile movements of all classes, divides them into four groups:—

(i.) Impulsive or spontaneous movements:—Comprising the stretching and yawning movements, noticeable as the infant wakes from sleep, also limb movements in the bath, protrusion of the lips in pouting. These are supposed to be due to spontaneous discharges of energy in the motor cells of the nervous system, and not to be due to the action of any external stimulus. As there be no evidence of a *mind* at this stage, they receive the name of "spontaneous" or "impulsive." It is in this class that most of the survival movements occur.

(ii.) Reflex movements:—Occurring as a result of some external or internal impression, such as stimulus of touch, sight, hearing, or visceral change. They appear at, or very

[1] Preyer, "The Mind of the Child": English Translation, "The Senses and the Will." Appleton, New York, p. 195, et. seq.

soon after, birth, and include the movements of breathing, swallowing, reflex movements of the eye in response to light, spreading of the toes in response to stroking or warming the soles of the feet.

(iii.) Instinctive movements:—These also require the stimulus of sense-impression to arouse them, but according to Preyer, differ from the purely reflex in being always preceded and accompanied by a distinct emotion. They are, therefore, accompanied by changes on the psychical plane. They all have a definite aim. He cites as instances:—pecking movements, by which a chick breaks the shell or seizes a fly, running or walking, biting, chewing, and so on; in fact any purposive movement which the individual does not voluntarily acquire for himself, but seems to inherit from his ancestors. Some of them appear at birth; others, such as walking, only at a later stage. We speak of teaching a child to walk as if walking were a purely voluntary and intelligent act, learned by imitation, it is true; but in fact, as we see below, we really here draw out by touch stimulation an instinctive movement. The rhythmical alternate movements of the feet before walking have been noted by Baldwin during the ninth month, *i.e.*, before the child can really balance itself on its feet. By holding a young baby on the palm of my hand, stomach downwards, and supporting the head with the other hand, and allowing the feet and hands to touch the counterpane of the bed, I have elicited alternate movements both in the feet and hands as early as the third day after birth (see fig. 4).

(iv.) Voluntary and Intelligent movements:—These are not inherited; each individual acquires them for himself. They are preceded by, or caused by, an "idea." They therefore not only have a definite purpose, but are consciously and intentionally performed. In the process of building up and elaborating of these intelligent movements, some of the most interesting problems in Mental Philosophy are involved. They include the origin of the will and of the intellect. Preyer, Baldwin,[1] and others have shown

[1] Baldwin, "Mental Development in the Child and in the Race," p. 81.

how important a part imitation and suggestion play in their growth. How many nerve processes occur in them is well shown by Preyer where he says: "In order to imitate, one must first perceive through the senses; secondly, have an idea of what is perceived; thirdly, execute a movement corresponding to the idea." He gives as instances a child making faces, such as pouting at a stranger or a nurse; holding out the arms to show he wants to be lifted up; speaking definite words. He considers walking to be simply an instinctive movement, not an intellectual or ideational one, because a child does not see others walking and then determines to imitate them, but because the parent or other instructor holds the child up by the arms or body, and makes its feet touch the floor, and thus sets in action the instinctive reflex of this mode of progression.

Such as classification of early infantile movements, however helpful to the psychologist, is unsatisfactory to the biologist. By bringing in the terms "consciousness," "intention" and "idea," even when we translate them into terms of their physiological correlatives of higher and lower cerebral and spinal changes, we get no nearer understanding their origin; while the true meaning of the intermediate stages through which the perfected forms of movement have passed, and by which alone they have been rendered possible, is entirely lost. In harmony with the method of enquiry that evolution has introduced, we need to study these movements in their gradual growth to perfection, not only in their ultimate forms.

Evolution of the Human Limbs.

Leaving then, for a while, the study of infantile movements along the lines of their mental accompaniment, *e.g.*, whether they are conscious, volitional, or reflex, let us study them along the lines of their possible evolution. The structure of the limbs in which the movements occur will be our first guide. A study of the limbs of animals shows that their structure varies according to their habits and instincts, and so directly or indirectly with the food the

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animal eats or the means by which it is acquired.[1] In this way limbs have been modified according to their use for *locomotion*, in water, land or air; *prehension*, tree and rock climbing; *destruction of prey*, carnivorous animals; or *manipulation*.

In the following paper I have confined myself mainly to the movements of the upper limbs, though movements of head and neck and legs present equally interesting problems.

The Structural Plan of the Human Hand.

In studying the general plan and structure of the human hand and comparing it with that of other animals we notice that while it is most highly developed in function, it is also in some respects the least modified in the general plan of its skeleton. In shape and bones it is more like the primitive amphibian paddle[2] than is the limb of any other mammal, even including the monkeys, and this fact alone debars us from looking upon the other existing mammals for traces of direct human ancestry.

It is also fully established that the primitive form of organic life, including that of man, was aquatic, and the hand must have existed as a paddle, at the time that gillslits in the neck were in functional activity. Von Bardeleben[3] shows the human hand possesses some very interesting traces of such formation. Finally, the method of development of the hand seems to indicate that the palm is originally the more important, and the independence of the fingers is acquired at a later stage of existence.

The more tempting and more varied land dietary, with its greater stored-up energy, must have had some share in alluring our ancestors from the sea and marshes, and in turn have helped in the transition from cold to warm-blooded

[1] For a full discussion of this question as it affects man, see Munro: "Prehistoric Problems," chapter ii., "On the Influence of the Erect Position," being the Presidential Address in Anthropology at the British Association at Nottingham, 1893.

[2] Romanes, "Darwin, and after Darwin," vol. i., pp. 54, 183.

[3] "Mammalian Hand and Foot," *Proc. Zoolog. Soc.*, London, April, 1894.

life. No doubt, too, the greater stored-up energy in the food helped in the capacity for further variation in structure and growth, and the higher land animals have been divided by Professor Owen into three classes of vegetable feeders: (1) root eaters, (2) plant eaters, (3) fruit eaters; and two classes of carnivorous animals: (1) insect eaters, and (2) flesh eaters. The habit of obtaining and devouring each different kind of food has resulted in the formation and fixation of a specially adapted limb for each purpose.

Darwin says:—"Our progenitors, no doubt, were arboreal in their habits and frequented some warm forest-clad land.[1] . . At a still earlier period the progenitors of man must have been aquatic in their habits, for morphology plainly tells us that our lungs consist of a modified swim-bladder which once served as a float. The clefts on the neck in the embryo show where the branchiæ once existed. . . . We apparently still retain traces of our primordial birthplace, a shore washed by the tides."

Analysis of Early Infantile Movements.

Let us now turn to the subject with which we are immediately concerned,—the study of the early movements of the human infant—especially those of the hand and arm. We shall see how readily the spontaneous movements group themselves in terms of their value in the racial struggle for existence and survival of the fittest. Limb movements to be of value must be appropriate; (i.) to simple progression, either aquatic, terrestrial, or aerial; (ii.) to prehension, or arboreal existence; (iii.) to manipulation, including the destruction and breaking up of food, or the search for it by digital investigation into nooks and crannies; and finally, to the making and using of tools or weapons.

Limb movements having been once acquired by the race, may persist in the individual in their fully developed form, or may remain only as partially developed rudiments or vestiges.

[1] "Descent of Man," pp. 160, 161.

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"Survival Movements" of Locomotion in marshy or aquatic surroundings.

FIG. 1.

Showing primary position of hands and digits. Second month of intra-uterine life. Also position of foot. From His.

FIG. 4.

Drawn from life, eighth day. Showing position of hands and digits, and toes when child suspended his own weight. Left thumb folded forward, but not in use.

If we watch carefully the position of the limbs of an infant either soon after birth or almost any time during the first three or four months, we find them sometimes in the primitive development position, viz.:—folded across the chest, thumb towards the head and with the palm towards the thorax; but much more often the palm is away from the chest-wall, and is directed anteriorly by means of extreme pronation, the dorsum of the hand often lying on or near the shoulder, sometimes an inch or two outside. As the child wakes up the elbows begin to open out and the palm is pushed outwards in a way that would be useful in locomotion, especially in a fluid or semi-fluid medium. In fact, it

FIG. 5.

Drawn from life, eighth day, to show position in which the child was held to draw out reflex movements of progression. Child supported body weight by legs, which showed marked alternate movements. Arms also showed alternate movements, though to a less extent.

is the movement of a paddle. Slow, rhythmical movements of flexion and extension of the fingers occur, which instead of possessing the quick, incisive character of voluntary movements partake of the sluggish rhythm so familiar to the visitor to the tanks of an aquarium. They often occur in a series of three at a time during a quarter of a minute, then follows a pause during which there is apparently an accumulation of energy in the nerve cells. Then another series of spontaneous discharges takes place, to be in its turn followed by another pause. It was this peculiar sluggish rhythm, so unlike that of any other limb movements that first suggested to me that they might be survival movements of aquatic or amphibian life.

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The resemblance becomes even greater when we further examine these movements. As the fingers become fully extended, a rotatory movement of the wrist—hyper-pronation—occurs, by which the ulnar border and little finger knuckle are lifted upwards as in raising a cup to the mouth, though the elbow-joint becomes extended instead of being flexed, the hand is thus moved backwards and outwards as a paddle would be used in swimming. The whole of this movement persists throughout life associated with the act of yawning, which act is thus found to be one of the most fundamental limb movements in the human being. It is also frequently met with in the blind where it appropriately serves the sense of touch.

It may be urged that such a movement or series of movements is not necessarily suggestive of aquatic progression, but may be the remains of a four-footed land progression. That this is not the whole case is proved, I think, by the fact that the movements in the two limbs are not always alternate but often synchronous, though alternative movements are clearly noticed when the child is held face downwards and the palms and soles touch something rough. These movements, moreover, do not take place in an antero-posterior plane, but in an oblique plane half-way between antero-posterior and transverse. Other vestigial movements which are prehensile and truly terrestrial occur in an antero-posterior plane. The movement in the oblique plane can be well studied in water lizards, where the use of the eversion of the ulnar border of the manus is clearly manifest. Lastly, the rhythm is the rhythm of cold-blooded aquatic or amphibian progression, and though perhaps suitable for a purely graminivorous or root-eating animal, is not suitable for one that has to pursue some of its food with any degree of rapidity.

In the discussion of the psychological nature of these early spontaneous movements, Bain[1] showed that they did not depend for their immediate causation upon any reflex stimulation from the outside, nor upon any internal emotion,

[1] "Senses and Intellect," p. 67.

but rather upon an abundance and exuberance of nervo-muscular energy; he suggested that in the greater or less endowment of such spontaneous energy, and in the greater or less sensitiveness to external impression, we have already in infancy some key to the fundamental difference in temperament exhibited by the man of action and the man of feeling.

Other impulsive movements, such as "rolling of the eyes," "twisting of the neck," &c., occur. Perhaps the eversion in some cases of talipes valgus is an instance of the loss of a (racially) late acquired arching and inversion of the foot in infancy, which Huxley considers a vestige of arboreal life. If the above explanation of these early movements, as vestiges of a previous stage is untrue, I do not know how else to explain them. They serve no definite purpose, unless we admit the teleological one, that they exist for the purpose of exercising certain groups of muscles and are thus pleasing to the child, though then we leave unexplained the fact that other series of movements equally simple are not thus exercised at this stage, such as movements of supination. It can hardly be that they are meaningless as well as purposeless. It seems more natural to look upon them as physiological or psychological vestiges whose fuller study will open up some hitherto closed chapters of man's history, and while they excite our curiosity, yet also kindle our wonder at the distance man has travelled in his upward journey.

Reflex and Instinctive Movements.

Let us now consider the movements which Preyer embraces under the terms "Reflex" and "Instinctive." These are, according to him, started by the stimulation of some sense organ from without, and do not arise from any spontaneous discharge of nerve cells from within the organism. They differ from the previous ones also in having a definite aim, while they resemble them in depending for their first manifestation upon an inherited nerve organisation or nexus. The difference between Reflex and

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Instinctive movements according to Preyer is that reflex ones are not preceded by consciousness, while the latter are preceded by consciousness in the form of an emotion, though this does not amount to a definite idea. Such a division is not very satisfactory and only applies somewhat loosely, and accordingly the two are in this paper considered together.

The earliest reflex movements that occur naturally after birth are apparently those of respiration. These seem to be set in motion by the stimulus of external cold acting on the skin and mucous membrane of the nose—sneezing. The sense of touch seems the most active and most early in

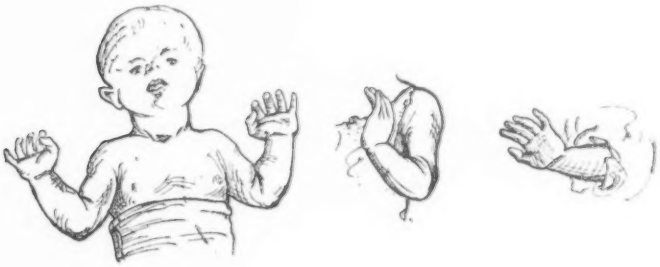

FIG. 3.

Drawn from life, on eighth day, to show several positions of hand at rest, with eversion of ulnar border and little finger; the last is a position useless for pure terrestrial locomotion. It is an aquatic "displacement position," though adaptable to arboreal life.

calling out reflex and instinctive movements. Then smell and taste, finally hearing and sight. With reference to the movements of the limbs, the sense of touch seems to be the sole avenue by which reflex movements are called into action. As before stated reflex movements of locomotion of both fore and hind limbs can be very early called into action by allowing the palms and the soles to be brought into contact with a rough blanket. These seem to be special applications of the stretching movements, and can only be studied when the child is wide awake. The stretching

movements are best noticed during gradual emergence from sleep.

Survival Movements of Prehension.

On touching or tickling the palm of the hand or sole of the foot, reflex action causes the fingers or toes to contract and to seize the object. If the right moment is chosen and especially if the rest of the body is not securely held, the clutching sometimes becomes so strong that the child can be lifted entirely from its bath or from the ground without any other hold. The grip seems even stronger on the ulnar side than on the radial border. How does such a movement fit in with our Developmental classification of the functions of

FIG. 2.

Further figures to show position of hand at rest in a child two months old. Power of supination very slight.

a limb into locomotion of varying kinds, viz.:—aquatic and terrestrial; prehension (arboreal locomotion), manipulation (movements involving supination and independence of the thumb, capacity for destruction and division of food, &c.)? They at once fall into the category of prehensile movements, and Dr. Louis Robinson in an article on "Darwinism in the Nursery," in the *Nineteenth Century* for November, 1891,[1] drew some very interesting parallels, and showed that by watching some sixty babies he found out that from the very beginning of their separate existence they possessed sufficient

[1] See also Dr. Robinson's paper on "Infantile Atavism," B.M.J., Dec. 5, 1891, p. 1226.

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grip and muscular power to sustain their own weight on a horizontal bar or finger, and even delighted in the process. I have myself frequently tried this experiment, but have had only varying success. I believe, however, that he is right in ascribing such early movements to inherited prehensile power, and that further investigation along these lines will be fruitful of other illustrations of the law of recapitulation.

In November, 1894, the *Nineteenth Century* published an article by S. S. Buckman on "Babies and Monkeys," in which further comparisons were instituted, from which the following extract is taken:—

"In the method of using its hands the baby shows to the full its descent from arboreal ancestors. When it wishes to take hold of anything, like a glass or a flower pot, it does not, like an adult, put the hand round it, or even put a thumb inside to use as a lever. On the contrary, it places all the fingers inside, makes no use of the thumb, and clasps the rim of the flower pot between the fingers and the palm of the hand. This is exactly the action which would be acquired from arboreal ancestors, in going from bough to bough they would take their hands, palm first, and would strike from above downwards, grasping the bough with the fingers. Such is the action of an infant picking up a cup. So little use have some monkeys made of the thumb, that abortion has resulted, and in the most arboreal species of monkeys known, the fingers have grown together, because the whole hand was used merely as a grasping hook."

Seizing or Clutching Movements, with a Psychical Accompaniment of Emotion or Idea.

Preyer discusses the Seizing movements under the class of Instinctive Movements, and says that the first noticed grasping at objects with manifest desire to have them was seen by Sigismund in a boy of nineteen weeks, and by himself in a girl in eighteenth week, and in a boy in seventeenth week. The factors in such an action are evidently very much more complicated than in the mere clutching of a stick. In the observations on my own child I paid more

attention to the method of grasping, and the date of the use of the thumb, and I extract the following from my notebook :—

Development of Independent Action of Thumb and power of opposing it to rest of the hand.

Eleventh week.—Baby distinctly uses his hands as a grasping organ to pull the feeding bottle towards him when very hungry, mainly by pressing ulnar border of palm on it and drawing towards him.

Twelfth week.—Does not use his thumb properly for grasping; when he tries to bring the bottle towards him he tucks the thumb out of the way. For the past fortnight, however, he has been beginning to grasp with his fingers, and will pull his mother's hair or dress if his fingers come in contact (see fig. 6, and refer to footnote).[1]

Fourteenth week.—Now moves his hand to press the teat into his mouth, and grasps things with greater vigour. No power of opposing the thumb to the rest of the hand, though it is noticed that the thumb does not get in the way so much as before. The thumb is also occasionally kept outside the fist.

Sixteenth week.—Has been using the thumb more and more since last note now, and never shuts it inside his fist, but always outside. He grasps objects intentionally now, but only for a few seconds. He does not hold out his hand to reach a thing but lets his finger travel over them as they lie on his lap; only after feeling them does he grip them.

Nineteenth week.—Played with a toy for the first time.

Twenty-fifth week.—Spontaneous purposeless movements of fingers now ceased. Movements of putting up and down and of flexion and extension of forearm.

Twenty-eighth week.—Grasping movements much more perfect, though he still does not pick up things between the tip of his thumb and finger. He occasionally grasps a thing

[1] On a Possible Obsolete Function of the Axillary and Pubic Hair Tufts, *Journal of Anatomy and Phys.*, 1892, p. 254.

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between the bent last joint of thumb and the fingers. In stretching out for a thing he often grips it between second and third finger, this especially if the fingers so slip round

FIG. 6.

Drawn from life, four months after birth, to show prehensile use of fingers, before the movements of the thumb are fully called out. The ball of the thumb in centre figure is used as a support. Inter-digital clutching movements of right hand figure illustrates a method of grasping objects of support which would be called into play in Dr. L. Robinson's theory of the use of axillary and pubic hair tufts (see reference in paper).

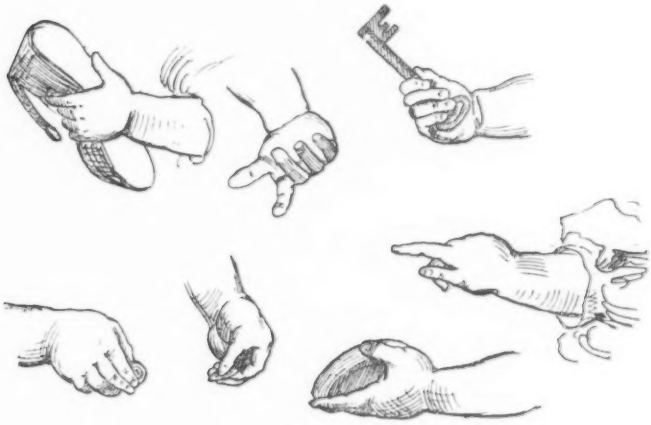

FIG. 7.

Eleven months. To show full development of powers of the thumb, of supination, and also the searching movements of index finger—digital investigation.

the object. In grasping small flat objects, such as a domino on the table, he grips between the tips of the fingers and the base of the palm, often bringing the ball of the thumb to

steady from the side. He now tries to pull things to pieces, such as a piece of paper (first appearance of purposive supination). The fingers are becoming more and more independent and have considerable power. Toes very rarely used for grasping, though they have not entirely lost that power. The thumb has been growing in importance and in variety of action very rapidly during the last week.

The reason I have quoted these notes in such full detail is to show how deeply rooted these inherited movements are, and by how slow a process those that do not survive disappear.

It is often six months or more before the more elaborate movements of manipulation possessed by the human being are rendered possible by the development of the capabilities of the thumb, and the power of purposive supination.

Survival Movements of Digital Investigation.

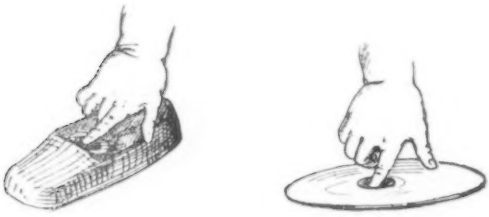

FIG. 8.

Drawn from a child twenty months old. To show full development of searching movements of index finger, a key in a shoe, and the hole in the head of a stethoscope.

Another survival movement presents itself prominently before our notice, namely, the way in which a child points and pokes at any object with its index finger. My wife and I noticed this digital exploration vaguely manifest about the seventh week, and it still persisted vigorously at the sixteenth month, though in a highly special and voluntary form. It is

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the method by which not only the form, but the resistance, the consistency, and texture of a thing are determined. The movement may have originated during arboreal life for burrowing into nooks and crannies, and to have owed its survival to the great knowledge it gives about food through the sense of touch. It has also very conveniently adapted itself to gesture language in pointing. A still more ultimate analysis may show it to be a specialised form of the stretching movements of aquatic life. In watching its early appearance, my own child at first indiscriminately used the second, third, or even fourth digit occasionally for this purpose, but finally rejected them all in favour of the index digit.

Lastly, I have frequently watched a child stroking the floor, the table, or other surface, and asked myself whether this originated as a movement of progression. In the movement of crawling the palms are flat downwards, fingers directly forwards. In this stroking movement the hands are pushed outwards away from the middle line, not inwards towards the centre, though when voluntary movements are performed there may be a to-and-fro movement. This may or may not be a survival from movement of aquatic locomotion, but its very early appearance suggests it to belong to the inherited movements and not to those voluntarily acquired.

In conclusion, I should like to urge the importance of further study of these early infantile movements, and to plead for their inclusion in any complete study of the development of the nervous system. Child Study is very young amongst us, but such a line of enquiry as I have suggested offers fruitful reward. Whatever the rightful explanation, there is certainly a gradual selection and a gradual supersedence of involuntary movements by voluntary ones, and these appear in a definite order, each successive batch being more complicated than its predecessor. The independent use of the thumb and the power of supination seem altogether voluntary and are of late appearance, and deserve separate consideration under the group of "manipulation." Medicine teaches us how these late arrivals are early departures, not only does the

power of supination and of opposing the thumb become early lost in various poisonings; but even the gait of the infant acquiring the art of walking reminds us irresistibly of that of the ataxic drunkard, whose alcohol has made him temporarily lose the complete co-ordination necessary for normal progression. While, however, a study of the processes of degradation and decay is apt to leave a feeling of depression and even despair, the study of the opening powers of the infantile mind fills us rather with joy and hope.

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HEMIANOPIA, WITH ESPECIAL REFERENCE TO ITS TRANSIENT VARIETIES.

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THE following paper, with the exception of a few alterations, some of which were rendered necessary by the subsequent section of the brain in one of the cases described, was presented in June, 1897, as my thesis for the degree of M.D. of Cambridge. The paper was originally intended to deal with several extraordinary cases of transient hemianopia, associated with unilateral convulsions, which I have had the good fortune to meet with, the disturbance of vision lasting often twenty-four hours or more. Such cases, so far as I am aware, have never hitherto been described, and, in my opinion, they go far to prove a theory of cortical representation of the macula, and that the macular region in the retina is really innervated on the same plan as the rest of the retina. Their close analogy with epilepsy on the one hand, and with migraine on the other, the association of hallucinations in the blind field with hemianopia, and other points of interest, have led me to extend the original title of my paper. I shall, therefore, deal with my subject under the following headings:

(1) Bi-temporal hemianopia.
(2) Bi-nasal hemianopia.
(3) Hemianopia in hysteria.
(4) Homonymous hemianopia.
(5) Quadrantic hemianopia.
(6) Colour hemianopia.

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(7) Hemianopic hallucinations.
(8) Hemianopia commencing with blindness.
(9) Central incomplete hemianopia.
(10) Double hemianopia.
(11) Cortical representation of the macula.
(12) Transient hemianopia.

I have to thank most of my late chiefs at the National Hospital for the Paralysed and Epileptic, Queen Square, Dr. Beevor, Dr. Bastian, Dr. Hughlings Jackson, Dr. Buzzard, Sir William Gowers, Dr. Ferrier, and Dr. James Taylor, for permission to make use of cases under their care in the hospital during my term of nearly two years as house physician. To Mr. Gunn also I am much indebted for his kind criticism and help, and to Dr. Luff for permission to refer to a case seen amongst his out-patients at St. Mary's Hospital.

Hemianopia, Monoyer's variation of the original word hemiopia, so as to indicate the symptom of half-blindness rather than half-vision, is, I think, preferable to the more clumsy term suggested by Hirschberg, hemianopsia. The term has generally been used to denote complete blindness in one or other half of the visual field, and it may be homonymous, in which either both the left or both the right halves of the two retinæ are anæsthetic, causing blindness of the opposite visual fields, or it may be bi-temporal, bi-nasal, or altitudinal. These latter forms will be only incidentally considered in this paper, altitudinal hemianopia, in which the dividing line is horizontal, occurring only in peripheral lesions of the optic path, as in neuro-retinitis, or possibly from disease of the chiasma, though, as suggested by Wilbrand (17), it is possible to imagine a loss of the lower quadrant on the one side and the upper quadrant on the other side, due to lesions in each cuneus. Indeed, such a case has now been recorded by Weymann (16), in a man who had a sudden fit, with loss of consciousness and hemiplegia. His vision was much affected, and perimeter charts showed the case to be one of double quadrantic hemianopia, the lower right and upper left quadrants in each eye being blind.

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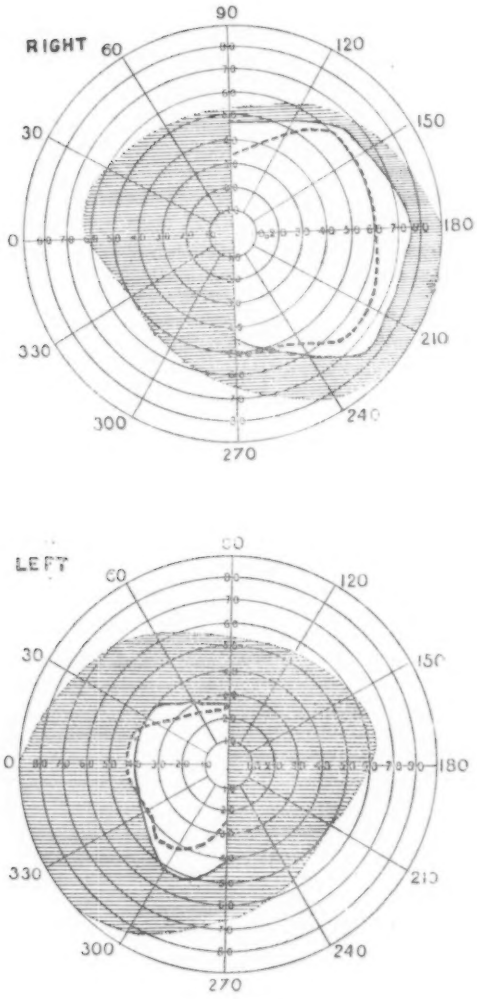
Bi-temporal Hemianopia.

The bi-temporal form can only be caused by a lesion at the chiasma, generally by a growth pressing down on its centre, as in tumours of the pituitary body, in acromegaly, or a fractured base. With both eyes open, the visual loss in these cases amounts to a bilateral peripheral contraction of the field down to 60°. Würdemann and Barnes (1) record an interesting case of bi-temporal hemianopia with the hemianopic pupil symptom, probably due to hæmorrhage in the region of the chiasma. The visual loss proceeded to nearly complete blindness, and then cleared up, a second attack later on causing right homonymous hemianopia.

Bi-nasal Hemianopia.

The bi-nasal form is the rarest, possibly because of the difficulty in examining for it, and the extremely slight visual loss when both eyes are open. It has been observed in tabes, and in one or two obscure cases probably due to a symmetrical neuritis of the outer parts of the chiasma, and it is possible to imagine symmetrical growths on both sides of the chiasma giving rise to it, as in Knapp's (5) case, quoted by Starr. Hamilton (2) has recently described and given perimeter charts of a case of bi-nasal hemianopia occurring in a man aged 56, who suffered from aortic regurgitation, and had two seizures followed by right hemiplegia. There was double optic atrophy, and considerable diminution of visual acuity, with partial colour loss in the remaining temporal fields, white and blue alone remaining. The bi-nasal hemianopia was, however, absolute, and was ascribed to embolism damaging the vascular supply to the tracts, both being symmetrically affected. No autopsy was obtained. The description of the case is, however, meagre, and it may possibly have been an instance of hemiplegia occurring in a case of tabes.

Strange to say, this form of hemianopia has also been recorded twice as occurring in hysteria, one case by Dr. D. B. Lees (3), in which bi-nasal hemianopia was found in a boy aged 11, and which had disappeared by the next day.



----- Blue field.

FIG. 1.

Bi-nasal hemianopia, after Hamilton.

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Another instance is figured by J. H. Lloyd (4), from a case of Mitchell and de Schweinitz, in which also the remaining half fields are much contracted.

Hemianopia in Hysteria.

Homonymous hemianopia, or blindness to one side, may rarely occur in hysteria. Briquet (7), as long ago as 1859, says, "In some hysterics either lateral half of the retina may be insensitive, giving rise to hemianopia," but he gives no method of examination. Svynos (12), Galezowski (9), Rosenthal (13), Westphal, and Bonnefoy (6), also give cases, Svynos mentioning that hysterical hemianopia is more frequent on the left side. Dercum (8) also gives the charts of a case of left hemianopia in hysteria with contraction of the remaining half fields. According to Galezowski and Daguenet (11), hysterical amblyopia sometimes assumes the form of hemianopia, or of a transitory central scotoma, or even of complete amaurosis of shorter or longer duration. This hysterical amaurosis is rare and affects sometimes one eye, sometimes both. Galezowski (10), quotes a case of a young woman who, being frightened by her sister being attacked with cholera, fainted, and on her recovery from the faint was quite blind. After some days her vision returned on one side, taking the form of right homonymous hemianopia. The blind field then also began to recover, her field of vision enlarging daily, and soon her cure was complete.

I have met with one similar case.

M. P., a married woman, aged 29, was admitted into the Queen Square Hospital, under Dr. Buzzard's care, in September, 1895. She had had numerous fits, probably hysterical, and when admitted she was quite unable to stand, or even to move the legs at all. There was general analgesia of the whole body, and tactile anæsthesia of the feet and legs up to the knees. There was no unilateral loss of the special senses, but on taking charts of the visual fields with the perimeter there was found to be left hemianopia, with contraction of the remaining half fields. Her vision was R. V. $= \frac{6}{36}$, L. V. $= \frac{6}{24}$, though on putting the trial frame before her eyes and a plain glass instead of a lens, her

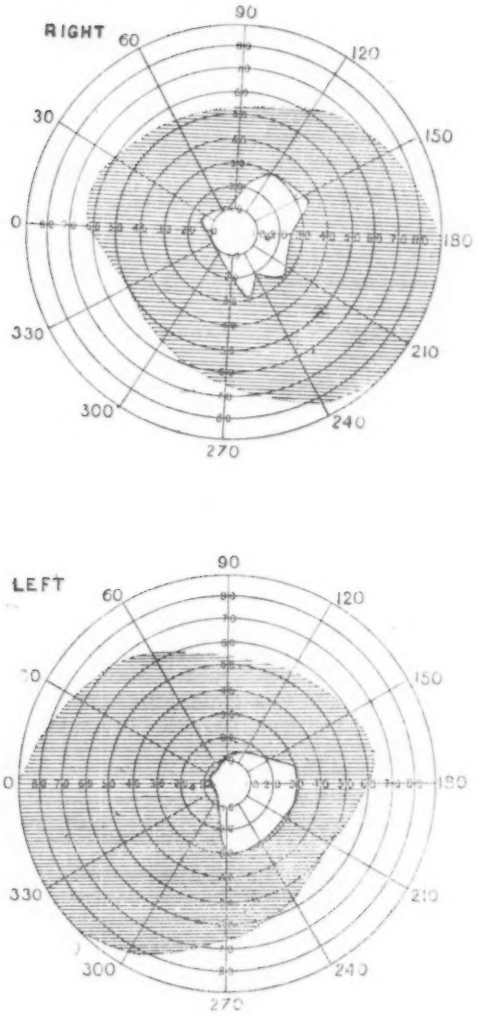

FIG. 2.

M. P.—Case of hysteria, with left hemianopia, and contracted fields.

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vision at once improved to R. V. $= \frac{6}{12}$, L. V. $= \frac{6}{9}$, nearly. A week later I suggested to her that she should not be able to see towards the right side, and the next morning when she woke up she was totally blind, remaining so all day. Her pupils reacted perfectly to light, but she was much upset at her loss of sight, and a match suddenly lit and brought quickly near the eyes produced no reflex starting or movement of the lids. In the evening the passage of galvanic shocks through her eyes and temples, combined with suggestion, restored her vision partially, and by the next day it was much improved. She steadily got well under treatment by the wire brush, the visual fields gradually enlarging, and acuity of vision improving, until her discharge a month later, when her visual fields were quite normal again, and vision $\frac{6}{5}$ in each eye.

Homonymous Hemianopia.

Homonymous hemianopia, the ordinary form, is generally due to a lesion in the optic path anywhere between the chiasma and the occipital cortex, either in the optic tract, the external geniculate body, pulvinar, the optic radiations in the posterior part of the internal capsule, or in the cortex of the cuneus.

A case has been reported by Eskridge (44), of right hemianopia, complete up to the mid-line, which was caused by a gun-shot wound, the *post-mortem* showing complete destruction of the left cuneus alone, as clean as though done for a physiological experiment. Henschen would limit the half-vision centre in the cuneus to the grey matter of the middle third of the lips of the calcarine fissure, on the internal surface of the posterior lobe. In the large majority of cases hemianopia is due to cortical softening, but various tests have been devised to distinguish between cases due to damage to the tract, or internal capsule, or to destruction of the cortex. The best known is the hemianopic pupil of Wernicke, the pupil contracting when light is thrown obliquely through it on the sound side of the retina, but remaining motionless when the light is thrown from the other side on the anæsthetic half of the retina. This symptom, if present in homonymous hemianopia, proves the lesion to affect the optic tract between the chiasma and

the ant. corpora quadrigemina where the fibres concerned in the pupillary reflex leave the tract to reach the nucleus of the third nerve. This symptom occurs in hemianopia due to tumours of the base or crus pressing on the tract, in the bi-temporal hemianopia due to lesions of the chiasma, acromegaly, &c. Homonymous hemianopia may also occur in tabes, as I have seen in two cases with optic atrophy, and in disseminated sclerosis also with optic atrophy, probably due to a patch of sclerosis affecting the optic tract.

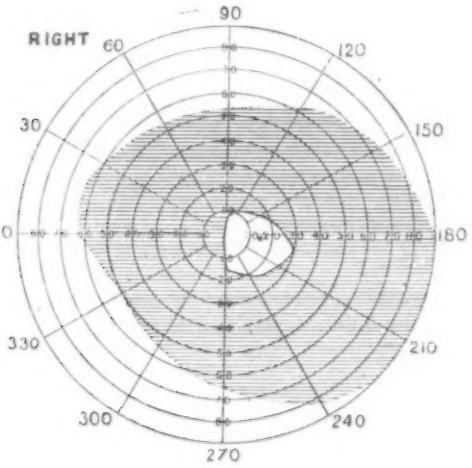

FIG. 3.

W. L.—Case of tabes, with optic atrophy. Left hemianopia, and contraction of remaining half field. The hemianopia had been noticed by the patient for at least eight months, but when the charts were taken the left eye had become blind.

If the pupil reacts equally well when the light is thrown from either side, the hemianopia is either functional, or due to a lesion in the optic radiations or cuneus.

Visual sensations are divided into the three perceptions of light, form, and colour, and various subdivisions of the occipital cortex have been allotted to each, Wilbrand placing the colour centre in front of the apical region, and Sir William Gowers accepts his theory. Mackay (18), however,

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proves that there is always some diminution of the form sense in so-called pure cases of hemiachromatopia, and according to Swanzy (45) "It is now generally believed that relative hemianopia (*e.g.*, colour hemianopia alone) is the result of a lesion of less intensity than that which causes absolute hemianopia, ... and that the colour sense is more easily affected by disease than the form or light senses, and that, too, irrespective of the position of the lesion in the visual path." Again, the light sense only may be retained, perception for both form and colour being lost.

Hemianopia, if occurring alone, is probably cortical in origin; but, according to Swanzy the chief diagnostic symptom of a cortical lesion is "vision nulle," that the patient, though he may be aware of the loss of half his visual field, is as unconscious of any scotoma as a healthy person is of his blind spot. Lesions below the cortex, as in the optic radiations, are said not to give rise to vision nulle, that the patient then has a sensation of darkness in the blind half field. I have examined for this symptom several times without any satisfactory result, and in one case to be presently related, in which the lesion was in the posterior part of the internal capsule, the patient was aware of no defect of vision whatever, and could with difficulty be persuaded that a considerable portion of his visual field was lost.

Quadrantic Hemianopia.

Quadrantic hemianopia is said to be generally due to a cortical lesion, the lower part of the cuneus corresponding to the lower quadrant of the field in each eye, as in a case published by Hun (15) in 1887. Wilbrand mentions the great preponderance in the statistics of cases in which the left lower quadrant is affected.

Sir W. Gowers (14) also figures a case of quadrantic hemianopia in which the diagnosis was a lesion near the hinder part of the thalamus. The following case is a good illustration of the fact that quadrantic hemiachromatopia may be due to a lesion in the posterior part of the internal capsule.

R. F., a man, aged 43, in August, 1896, was suddenly seized with left hemiplegia without loss of consciousness, with the sensation as though he had been struck on the back of the neck. The hemiplegia was not complete at first, but became nearly so later, with great rigidity of the arm and leg, and pain on movement. When admitted into the Queen Square Hospital in March, 1897, under Dr. Bastian's care he had almost complete left hemiplegia with great rigidity, partial left hemianæsthesia,

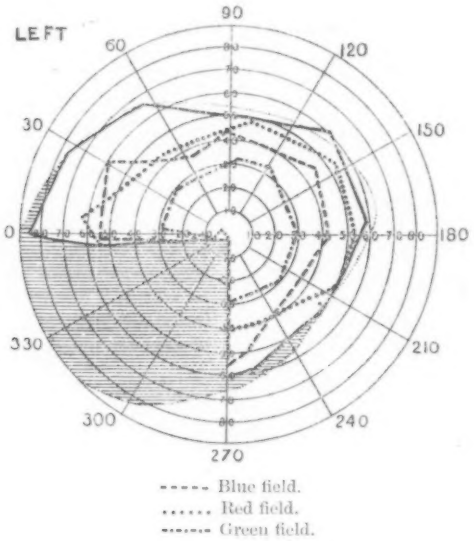

FIG. 4.

R. F.—Case of left quadrantic hemianopia, complete for colour, but partial only for form sense. The field of the right eye could not be charted by the perimeter, owing to the patient becoming exhausted by sitting up. Careful testing, however, showed it to correspond accurately with that of the left eye.

and anæsthesia dolorosa most marked on the left arm. He had evidently some affection of his left field of vision on rough testing, and on being carefully tested with the perimeter he was found to have complete loss of all colour perception, even of large area, in the left lower quadrant for each eye; small objects also, as the head of a pin, or a disc of white paper 1 cm. in diameter, were

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also invisible, and he could not distinguish between one and two fingers held up in the same area, though he could readily do so in each of the remaining quadrants. A large object, as a hand moving, or a sheet of notepaper, he could see readily enough in the affected area, and his light perception was also good. His discs and fundi were normal, and he was not aware of any defect of vision before being tested. His heart, rapid from the first, became very irregular a month after admission, and a loud systolic murmur appeared. He became much weaker and delirious, and died suddenly on April 14. At the autopsy, eighteen hours after death, the cuneus was found quite normal on each side, but there was a patch of softening in the right internal capsule. The heart and valves were normal.

Hemianopic Hallucinations.

Hallucinations in the blind field are said by Swanzy (19) to be possible in lesions of the optic radiations, but not in hemianopia due to cortical damage. The reverse I think rather is more common, and the only case with autopsy I can find which supports his contention is one published by De Schweinitz (27), of a man, aged 29, who had visual hallucinations of chairs, tables, and other furniture, to his left, followed soon after by convulsions and left hemianopia. He had repeated seizures, and developed delusions of grandeur, and was sent to an asylum. At the autopsy made by Dercum, gummatous infiltration of the base of the brain, pressing on the right optic tract was found but, no mention of the occipital lobe is made. Henschen (24) records four cases, two with autopsy. In one case of right hemiplegia and hemianopia with hallucinations of seeing a boy near the foot of the bed on the blind side, the autopsy showed softening in the occipital lobe and internal capsule. In the other there was left hemianopia without hemiplegia, and hallucinations of seeing persons and faces in the blind field, and the autopsy showed softening in the right cuneus and lingual lobe, and in the right thalamus. Mackay (18) records the case of a farm labourer, aged 37, who suffered from partial right hemianopia following a blow on the back of the head, with recurring attacks of erythropia, and of

visual hallucinations, lasting a few minutes, of folk and horses moving in a reddish atmosphere, limited to the blind field and never crossing the mid-line. He had complete right hemiachromatopia with some impairment of the form sense, the dividing line passing vertically through the fixation point in each eye. He died three years after the injury, being seized with right hemiplegia and aphasia, but no autopsy was made. Mantle (25) describes the case of an artist, who had pure motor aphasia, followed eleven weeks later by word deafness, and word and mind blindness. He became gradually deaf, with dimness of sight, the deafness and blindness becoming complete in about twenty-four hours, though the pupils still reacted to light. The deafness and blindness continued for ten days, when his vision gradually cleared up into right hemianopia after a fortnight. He had two relapses of loss of sight, the second one lasting four weeks, but clearing up again into right hemianopia. He also had visual hallucinations in the blind field of a man standing at the back of his head, holding two lighted candles. Seguin (28) in 1886, was the first to record a case of this kind, and others have been recorded since by Bidon (20) in 1891, Putzel in 1888 (26a) and by Wilbrand (17) in 1890. In Wilbrand's case there were hallucinations of heads, furniture, cats, &c., in the blind field, and at the autopsy there was found hæmorrhagic softening of the medullary substance in the third occipital convolution, the superficial cortex being yellowish and discoloured. Dr. W. S. Colman (21) has recorded the case of a woman, aged 45, suffering from left hemiplegia, hemianæsthesia, and hemianopia, due probably to a lesion in the posterior part of the right internal capsule. For three weeks after the onset she saw numerous faces of men, women, and children, always in the blind field only. The faces were unfamiliar and not unpleasant, and she quite recognised that they were unreal. The hemianopia persisted, although the hallucinations passed off. Peterson (26) thinks the pathological basis in these cases must be irritation of the cortex of the occipital lobe, and he describes hallucinations occurring only in the right half fields in a case of chronic delusional insanity

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without hemianopia, and mentions a case of Dr. J. Van Duyn's, of Syracuse, U.S.A., of a woman, aged 45, who developed left hemianopia, and one week later had constant hallucinations of seeing animals and children moving in the blind fields, this continuing for four weeks, and then suddenly ceasing, though the hemianopia persisted.

In Putzel's case, a man, aged 54, developed right hemianopia, followed ten days later by hallucinations in the blind field of men, flies, insects, &c. At first he recognised their unreality, but after a few days he became convinced they were real. The spectra became more frequent, and he would then hunt for them in cupboards and corners. He developed uræmia and died six weeks from the onset of the hemianopia. At the autopsy a spot of softening was found in the left cuneus, extending across the calcarine fissure, and forwards to the parieto-occipital fissure. The kidneys were extremely cirrhotic.

Hack Tuke (22), mentions hemianopic hallucinations as occurring "in the sane as well as in those subject to epilepsy, migraine, anæmia, and delusional insanity. When coincident with hemianopia, such hallucinations probably originate in a cortical irritation or malnutrition limited to the occipital lobes." Dr. Colman, in describing some hemianopic hallucinations, mentions that they consist usually of faces, the most easily remembered individual characteristics, or else of objects, such as rats, beetles, &c., which from early infancy are usually associated with sensations of alarm. He agrees in the refutation of Brewster's theory of hallucinations, that there is an actual excitation and chemical change produced in the peripheral organ, such as the retina, as would have been produced had an image of the real object fallen on it. He goes on to say that it is due evidently to a condition of the perceptive centres, and draws attention to the fact that opacities of the vitreous may at first cause muscae volitantes, which are recognised to be part of the individual, though later, as the mental condition deteriorates, they may take the form of faces or animals. These are for a time recognised as existing only in the patient's own sensations, without any external warrant,

but later on he fails to recognise that they have anything to do with himself, and refers them to the world outside him.

Binet has supported the theory of peripheral origin of hallucinations, and has shown that in hypnotic hallucinations the vision may be doubled by means of a prism before one eye, but this point is disputed by Hack Tuke. The truth would appear to lie midway between the two assertions, and we may regard hallucinations and illusions as two extremes of the same phenomenon, and that there is no hard and fast line separating the one from the other. On this basis, we can understand how that in a patient, hypnotised to believe that a spot on a piece of paper is a photograph, the prism test may produce doubling of the images. This test, then, depends on the strength of the external stimulus, and when the vision is more subjective in origin, and persists after closure of the eyes (approaching Esquirol's definition of an hallucination as differing from an illusion in that, in the latter an external object forms the basis of an erroneous perception) then lateral pressure on the eyeball would cease to cause doubling of the image.

J. Christian (43), in his article on hallucinations, adopts Ritti's theory in placing the seat of hallucinations in the optic thalami, and he lays down in his fourth law that there is always the reminiscence of something previously seen, that the sensory centres create nothing, invent nothing. This axiom will, I think, hold good if not pressed too far, that is to say, the main outlines of figures appearing in hallucinations must have been perceived before, though the faces may be unfamiliar, the figures differently grouped, clothed, &c. Talma, the actor, had the power of voluntarily producing the hallucination of divesting his audience of clothes, hair, flesh, &c., and of seeing them as skeletons filling the theatre, and applauding. That he must have seen skeletons at different times of his life may easily be believed, but that he could have seen so many at one time, and grouped in such a way, is impossible.

Assuming then that visual hallucinations occurring in the blind field are not peripheral, that is, retinal in origin, the question arises as to the seat of their production.

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Peterson considers them due to irritation of the occipital cortex. In de Schweinitz's case, however, already mentioned, the lesion was in the optic tract, and occasionally hemiopic hallucinations have been met with without hemianopia, as in delusional insanity. It is, in my opinion, impossible to conceive that such highly elaborated sensations as have been described in cases of hemianopic hallucinations, such as visions of persons, animals, furniture, and the like, can possibly be elaborated in the half vision centre in the cuneus. This centre, as a sensory centre, no doubt corresponds to Hughlings Jackson's "middle level" of the motor representation of the limbs and trunk, the so-called "motor area" in the Rolandic cortex. Low-elaborated visual sensations, such as red and green lights, or the fortification spectra of migraine, are probably originated by a paroxysmal discharging lesion in the cuneus, but complex visual phenomena are probably no more originated there than are complex purposive movements originated in the "motor" limb centres. A higher visual centre, then, must have a large share in their production, and to this centre, which possibly is the angular gyrus, we must look for the ultimate seat of elaboration of such sensations, though such action may be set up reflexly by peripheral irritation of the optic path. For this purpose we may consider the cuneus to be part of the periphery, and in this way we are enabled to understand how complex visual hallucinations may be due to disease anywhere in the optic path, either to vitreous opacities, retinal disease, lesions of the optic tract, optic radiations, or cuneus. The peripheral disease merely determines the localization of the spectra, which in disease of one eye are referred only to the visual field of that eye, whereas in disease of the optic tract, optic radiations, or cuneus, they are referred to the corresponding half fields.

An instance somewhat parallel to migraine, of discharge in a sensory centre, is seen in some cases of trigeminal neuralgia, rightly called epileptiform neuralgia, in which the discharge occurs, no doubt, in a centre higher than the Gasserian ganglion, probably in the sensory nucleus of the fifth nerve, but which may be set up reflexly by irritation of a peripheral portion of the nerve.

I have myself met with five cases of hemianopia in which there were or had been visual hallucinations in the blind field.

In a case I published last year (23), of localized convulsions on one side, the patient on several occasions soon after the onset of his hemiplegia and L. hemianopia, which occurred six months before his admission into the Queen Square Hospital, had hallucinations in which he saw Buffalo Bill's camp, the men and horses &c., and fancied he heard the guns firing and the shouting at the exhibition at which he had worked ten years previously. These hallucinations occurred at night on going to bed, but if he put out the light and turned over on his right side they disappeared, an evidence of the influence of peripheral stimulation by the light. Later on he had frequent attacks of seeing red and green lights straight in front of him ; in his case there was complete loss of the form and colour sense, but the light sense was fair, even out to the periphery. The autopsy showed cortical softening both of the cuneus and of the angular gyrus on the right side, but the internal capsule was normal.

The following case is an instance of partial hemianopia in which a peripheral zone of the left lower quadrant remained intact. This was in all probability due to softening of the right cuneus, and the patient also suffered from frequent attacks of seeing red and green lights, always to the left, in the blind field.

J. B., a woman, aged 36, with a history of probable syphilis, suddenly lost her sight ten years ago, the day after a severe flooding. At first she could see scarcely anything, but in a month's time when she went to a hospital this had cleared up into left hemianopia, which has persisted ever since. She had no hemiplegia at that time, but two years ago she had a slight attack of right hemiplegia ; and again in September, 1896, had an attack of left hemiplegia without loss of consciousness, but no further affection of vision. When admitted into the Queen Square Hospital under Dr. Beevor's care five days later, she had complete left hemianopia, total paralysis of the left leg and upper arm, but could move the left thumb, fingers, and wrist slightly. There was marked early rigidity of the left arm and leg, with slight left hemianæsthesia, but no affection of hearing, smell, or taste. The heart was enlarged, with a systolic murmur at the

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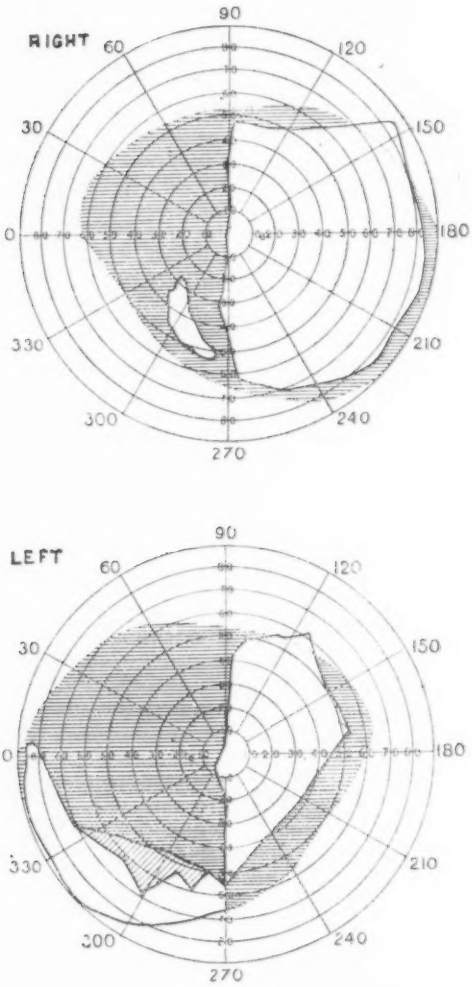

FIG. 5.

J. B.—Left hemianopia, complete in upper quadrants, but with peripheral strip of normal vision in lower quadrant, best marked in the left eye.

apex: artery walls thick, and albumen in urine $\frac{1}{12}$. The right arm and leg were normal, but her articulation was very thick and difficult to understand, and remained so for the six months she was under observation, this being indeed her only pseudo-bulbar symptom, as she could protrude her tongue and swallow quite well. Thickness of articulation is, I believe, an important symptom in double hemiplegia, and is often well-marked when there are no other bulbar symptoms proper.

By the end of October she had regained some vision in the periphery of the left half fields, and charts taken early in November showed that this return of vision was limited to the periphery of the left lower quadrant in each eye, a strip of about 20° in width from the extreme periphery inwards on the left side having regained normal vision, though the whole of the left upper quadrant and the central portion of the lower quadrant remained completely blind in each eye. In this peripheral strip in the left eye, she could distinguish between one and two fingers held up, and was able to recognise all colours, even in the extreme periphery, if of sufficient area, coloured cards about three inches square being generally used at a distance of about fourteen inches. A small corresponding patch in the lower nasal field of the right eye had also regained the light sense, but form and colour were not recognised in it. She remained under observation for five months longer, but the visual fields remained the same. During this time she had three fits, with loss of consciousness and left-sided convulsions, followed by temporary increase in thickness of articulation, but no alteration in vision. In one of these fits she dislocated her lower jaw. On several occasions she complained of feeling unwell, with headache, and of seeing red and green lights to her left. These were limited to the left or blind half field, never crossing the mid-line, and they appeared to be moving in both directions, from the periphery towards the centre and also in the reverse direction. They also seemed visible over the area of the peripheral strip of vision in the left lower quadrant.

The persistence of a strip of peripheral vision in hemianopia is rare. Delépine (30) records a somewhat similar case with autopsy, in a man who had sudden onset of right hemianopia with slight right hemiplegia. When his visual fields were charted, eight months later, there was found complete right hemianopia with the fixation point included

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in the seeing half, but with a strip of peripheral vision, about 10° in width, situated chiefly in the lower right quadrant of the field, and only in the right eye, the nasal field of the left eye being completely blind. At the autopsy, three months later, there was found softening of the left cuneus, with the exception of its postero-superior margin. This case, then, is not in accordance with that previously recorded by Hun (15) in which loss of the lower quadrant was found to be due to softening of the lower part of the cuneus only. They would, however, correspond if Henschen's view be accepted, that the upper edge of the calcarine fissure represents the upper quadrants of the retinæ, and therefore the lower quadrants of the field, the lower edge of the calcarine fissure representing the lower quadrants of the retina. In Delépine's case there was no peripheral vision in the nasal half of the left eye, and my case resembles it in so far that when first tested she seemed also to have no patch of peripheral vision in the nasal field of the right eye corresponding to the peripheral strip in the left eye. Careful repeated testing was necessary before it was discovered, and even then the visual acuity amounted only to seeing an object move, and no colour could be perceived in it, though colours were easily recognised in the periphery by the left eye. Delépine gives a reference (33) to another similar case.

Another case of hemianopia with hallucinations in the blind field I have had the opportunity of examining is that of a man, N. B., aged 51, who had an attack in June, 1896, in which he suddenly lost power of speech, using wrong words, and forgetting the names of things. At the same time the fingers of his right hand became numb and useless. This all passed off in two minutes. He then began to suffer from left-sided headaches, and during the next six weeks he had five or six similar attacks. During one of these subsequent attacks of temporary aphasia and weakness of his right side he suddenly noticed while reading that his sight was confused and that the print seemed to run together. After that he noticed he could not see so well to the right, and he used to bump up against things on his right side, and had to be careful whilst crossing the road. He also has had visual hallu-

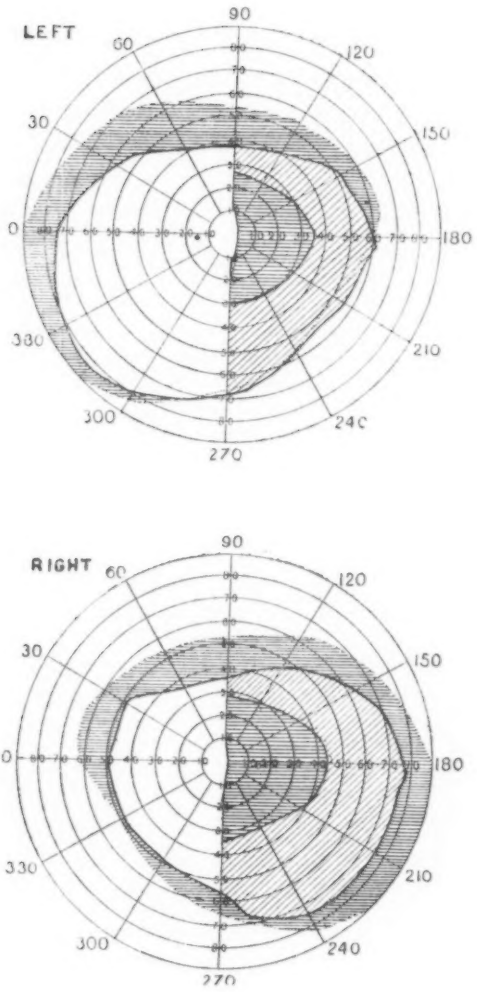

FIG. 6.

N. B.—Case of right central incomplete hemianopia. Fields for white spot 1 cm. in diameter. The area of indistinct vision is indicated by lighter shading.

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cinations of animals and faces moving about to his right. His vision has remained the same ever since, and when admitted into the Queen Square Hospital under Dr. Hughlings Jackson, in February, 1897, perimeter charts taken showed incomplete right hemianopia, in which the central portions of the half fields were completely blind over an area extending about 30° above and below the fixation point, and 38° laterally in the left eye and 45° laterally in the right eye. To the right of this blind area he could recognise the movement of even small objects, though his form sense was much impaired, and he could not distinguish between one and two fingers; he could, however, recognise colours even of small area. His condition while under observation has remained the same, there being slight weakness of the right arm and leg. When last seen in May he had had no further fit and was in good health, but the hemianopia remained exactly as before. There was no history of syphilis, and an accurate diagnosis seemed impossible, though cortical softening seemed probable.

I have met with two other cases of visual hallucinations on the hemianopic side, which will be related presently in full, one in a woman aged 51, who had partial left hemiplegia and a history of recurring convulsions. Her visual fields were normal, except once after a fit when I found absolute left hemianopia lasting from four to six hours. Later on, after another left-sided fit without hemianopia, she developed visual hallucinations in which she saw people in wheel-chairs, coloured brightly in green and gold, passing her always from her left side as far as the mid-line. In another case, a man, aged 54, suffering from partial right hemianopia and word blindness, had recurring convulsions. Some of these attacks appeared to be truly epileptic, but two have been typically Jacksonian, without loss of consciousness, and with clonic convulsions of the right arm and leg lasting for two hours. He frequently sees the faces of his children, his pet dog putting up his paws on to the bed, or his cat, sometimes school children running along, some being dressed in red cloaks as they used to pass his window at home, and similar objects in his blind field to his right. These hallucinations last only a very short time, but are frequent, and irrespective of the fits, and are suffi-

ciently distinct for him unconsciously to step to one side to avoid them, though he at once recognises their unreality. The case is an obscure one, but the lesion is probably cortical, possibly in the neighbourhood of the angular gyrus, and damaging the underlying optic radiations on the left side.

Hemianopia commencing with Blindness.

Hemianopia may commence with complete blindness, which, after a varying interval, clears up, leaving normal vision in the half fields of one side. This transient amaurosis must, I think, be due to inhibition of the remaining half vision centre, and is perhaps more liable to occur when the lesion is sudden damage to one cuneus. Dr. Allen Sturge (29) describes a most instructive case of absolute blindness coming on suddenly in association with right hemiplegia, hemi-anæsthesia, and loss of taste, smell, and hearing on the right side. On the next day some vision had returned in the left eye. Two days later the right eye was still quite blind, and there was complete loss of the right field in the left eye, but the left half field was quite good. The left half field in the right eye rapidly recovered vision, leaving right hemianopia, which persisted. This case is generally misquoted as one of hysterical hemianopia, because it was related during a discussion on hysterical hemi-anæsthesia at the British Medical Association meeting at Cambridge, in 1880.

Gowers (14) mentions a case in which complete right hemianopia cleared up to quadrantic hemianopia. In this case a partial lesion may have temporarily damaged the whole half vision centre, or possibly damage to the optic radiations may have cleared up partially, leaving the fibres of one sector destroyed.

The disturbance of vision does not often amount to absolute anaurosis, but frequently there is very great disturbance of vision at the commencement, the patient stating that he could see scarcely anything, and the vision gradually improves, leaving hemianopia on one side.

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The case described by Mantle (25) has been already related (see p. 14) of a man in whom right hemianopia was ushered in by complete blindness lasting twenty-four hours, during which the pupils re-acted perfectly to light.

Central Incomplete Hemianopia.

According to Gowers (14b), "when a complete hemianopia lessens, it may do so from the medial region towards the periphery so as to leave a symmetrical peripheral loss; or else from above or below, so as to leave a quadrantic defect. It rarely clears from the periphery, so as to leave symmetrical scotomata having one extremity in the central region."

Lang and Fitzgerald (31) have recorded such a case associated in its onset with hemiplegia and paralysis of the upward and downward movement of the eyes and lids, due probably to a lesion in the basal ganglia on one side. The ocular paralysis disappeared as the hemianopia cleared up from the periphery a week later, leaving central scotomata close up to the fixation point. They also give references (32) to several other cases. Wilbrand (17) also records one. Delépine's and Hun's cases have already been quoted, and the two cases I have met with—a woman, E. B., and a man, N. B., have already been related.

Double Hemianopia.

Double hemianopia also almost constantly commences with complete blindness, and according to Gowers it necessarily causes permanent amaurosis. Numerous cases are, however, now recorded in which complete blindness has suddenly occurred, or in which hemianopia on one side has been followed later by sudden complete blindness, and afterwards a small central area of vision returns, generally with considerably diminished acuity. The fields in these cases resemble the pin-point fields in hysteria, but a characteristic loss of power of orientation, and of finding their way about, is mentioned by Foerster (35), Groenouw (35b), and Vorster (35c), though it was not present in

Schweigger's case (35d), and this would distinguish them from the functional cases which seem in no way the worse for their restricted fields. In the latter case no doubt the half vision centres do perceive, and so visual impressions may produce reflex effects, though higher psychical influences inhibit the sense perceptions. A case of double hemianopia with autopsy is recorded by Schmidt-Rimpler (35a).

Cortical Representation of the Macula.

Such cases of double hemianopia, with return or persistence of central vision, are a very strong argument in favour of Foerster's theory of a cortical representation of the macula. A case recorded by Wilbrand (17), forms a converse to them : a man suddenly finding his vision confused, and on examination was found to have symmetrical scotomata touching the fixation point on the right side of each field, and equal in size to twice the diameter of the optic disc. A somewhat similar case I have myself met with has already been related. (See case of N. B., fig. 6.)

There are two opposing theories with regard to the representation of this region in the cortex. (1) That the whole of each macula is represented in each half vision centre, and that there is a special decussation of the macular fibres at the chiasma. This theory necessitates that the dividing line between the seeing and the blind fields in all cases of hemianopia, whether due to cortical or to tract lesions, should invariably pass round the fixation point, which is then included in the seeing half. Cases in which the line appears to pass close to or even to go through the fixation point are accounted for by individual variations in the nervous supply of the macula. Gowers strongly upholds this theory, and is of opinion that in all cases, if carefully and accurately tested, the dividing line would be found to pass around the fixation point.

Allen Starr (5) says " In no case of hemianopia is direct vision at the fixation point affected." Foerster's theory, quoted by Swanzy (19), supposes that the nervous supply of the macula is invariably arranged on the same plan as the

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rest of the retina, that each half of it is innervated from the corresponding half vision centre in the cuneus. Further, that there is a special area in each half vision centre in which the corresponding halves of each macula are represented, and which is more richly supplied with blood vessels, or at all events offers more resistance to vascular softening than the rest of the visual centre. According to this theory, in a total destruction of one half vision centre, as by a hæmorrhage, or in destruction of one optic tract, the dividing line between the seeing and blind fields would pass through the fixation point, but that in a lesion such as softening from embolism or thrombosis, this special region, owing to numerous anastomoses in its blood supply, escapes, and the dividing line would then pass around the fixation point, leaving it in the seeing half. This theory, therefore, will account for both varieties in the position of the dividing line in hemianopia, and, since both varieties are well supported by numerous careful observations, it is worthy of greater credence. Wilbrand (17) states that in 77 cases of lateral hemianopia the dividing line was central in 29, but of 32 cases of bi-temporal hemianopia in only 9 was the dividing line central. Cases of double hemianopia with persistence of a small central area of vision strongly support the latter theory that in each half vision centre the area representing the macula is less liable to suffer than the rest of the cortical centre. Complete proof that the macula is innervated on the same plan as the rest of the retina, and that there is not a special decussation of the macular fibres, is in my opinion afforded by the study of cases of transient hemianopia. In nine instances of this, occurring in three patients, I have examined this particular point as to the position of the dividing line with especial care, not trusting to the perimeter alone, which is not sufficiently accurate for such a delicate observation, but using a method as follows which was shown to me by Sir William Gowers. The patient is made to sit facing the observer with one eye shut, at a distance of about two feet. The physician then closes his own eye opposite to that shut by the patient, who is then told to gaze steadily into the observer's open eye opposite,

who in turn watches the patient's pupil. The line of fixation is thus the same in each case and the slightest wavering of the patient's eye is at once noticed and allowed for by the observer. Then, using his own visual field as the normal, and holding the test object, such as a small piece of white paper on the end of a black pen, half way between himself and the patient's eye, the latter's visual field can be approximately charted. It is, however, for determining the exact position of the dividing line in hemianopia that I find this method specially useful. A very small test object must be used, such as a piece of white paper 1 mm. in diameter, fixed on the end of a black pen and held close to the observer's face so as to be in the same focus for the patient as the observer's eye that he is looking at. If it is then brought slowly inwards towards the fixation line, it can be exactly determined by the observer, after repeated trials, whether or not the object is seen by the patient before it reaches the line of fixation. In nine instances of transient hemianopia, each lasting several hours, in which I have examined this point during the stage of absolute loss of half vision, I have found the dividing line to pass accurately through the line of fixation, no object, not even the flame of a match being perceived until it crosses this line. In these cases, since the vision clears up to normal in about twenty-four hours, more or less in different cases, the loss of function of the half vision centre must be due to exhaustion, and not to any gross lesion, and therefore the centre for the macula may suffer equally with the rest of the cortical visual centre. In all these cases, too, vision clears up from the centre to the periphery, central vision being first established, and the field gradually widening, an additional evidence that the cortical centre for central vision has greater recuperative power than that for peripheral vision.

On the other hand, in no case of persistent hemianopia that I have as yet examined by this method has the dividing line passed through the fixation point, though in some cases it passes extremely close to it, and I think it highly probable that the fixation point would be found included in the seeing half in all cases of persistent hemianopia, as Sir W. Gowers

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has suggested, if tested by this method. This apparent discrepancy between cases of transient and persistent hemianopia may, I think, be accounted for in one of two ways: (1) in cases of persistent hemianopia the cortical centre for the macula may have either escaped destruction, or have regained some of its functions; or, (2) the patient may have developed by education a new fixation point in his retina close to the original fovea centralis. This is, I think, most likely the case in those cases in which the dividing line passes very close to the fixation point. On the other hand, in cases of complete transient hemianopia there is no time for possible education of a new fixation point, and so the dividing line in them invariably passes through it.

Transient Hemianopia.

I now come to the main object of this paper, the discussion of *Transient Hemianopia*. This is a not uncommon phenomenon of migraine, and according to Gowers it sometimes occurs, like other manifestations of that disease, as an isolated symptom, apart from headache. Gowers (39) also, as long ago as 1877, mentions its occurrence in acute cerebral lesions with hemiplegia, during the stage of conjugate deviation of the eyes, as shown by the absence of any palpebral reflex to a finger darted at the eyes from the hemiplegic side. Swanzy (19), under similar circumstances, has seen it last three weeks, and in the following case I noted the presence of partial hemianopia four weeks after the attack of hemiplegia, though in another week it had disappeared.

E. W., aged 63, in September 1896, had a sudden attack of left hemiplegia, with partial loss of consciousness, and some interference with speech. On her admission to the Queen Square Hospital four weeks later, under Dr. Jackson's care, there was almost complete left hemiplegia, marked left hemianæsthesia, and partial left hemianopia, but no affection of taste, hearing, or smell. There was absence of any palpebral reflex to a finger darted at the eyes from the left side, though a brisk one was obtained from the other side. She could not count fingers further out than 30° on

the left side, and when reading the test types she would miss the left hand letter or the left half of a word. Vision was R.V. $= \frac{6}{18}$, L.V. $= \frac{6}{24}$. In another week her left visual field had quite recovered, though otherwise she remained much the same until her discharge.

Hemianopia, when due to an organic lesion, is rarely recovered from, and the following case is worth recording on account of the great improvement in the left visual fields which had been completely blind:

G. D., a man, aged 64, had an apoplectic attack on Nov. 3, 1896, with convulsion of the left arm and weakness of the left side, without loss of consciousness. At the same time he lost the feeling on the left side, and also the sight in the "left eye." There was no onset of complete blindness, and he could recognise people next day. When admitted into the Queen Square Hospital under Dr. Bastian, on Dec. 29, he had marked left hemiplegia, with rigidity and pain on movement. He could just move the left shoulder but not the elbow or hand. He could move his leg about a little, but could not stand at all, falling over at once to his left. There was definite slight left hemianæsthesia, and complete left hemianopia for all forms nearly up to the mid-line, but not including the fixation point. Vision was $\frac{6}{24}$ in each eye. Refraction, 2 D hypermetropia in each eye. No other affection of the special senses. His colour fields could not be tested owing to his being congenitally colour-blind. There were marked gouty changes in his left wrist joint and right foot, with visible chalk stones, and tophi in his right pinna. The arteries were much thickened, and there was a trace of albumen in the urine. The most probable diagnosis seemed to be a hæmorrhage in the posterior part of the internal capsule on the right side. After he had been in hospital three weeks, it was found that the hemianopia was not complete. He still did not flinch from an object darted at his eyes from the left, but he could tell when a large object as a hand was moving at 30° from the centre, though a light was not perceived until it had reached an angle of 45°, and he could not count fingers at all to the left of the mid-line. Two months after admission his left visual field had improved further, and he could tell when a large object was moving at 80° to his left, and could see fingers moving at 45° and count them at 30°, but in reading he still often missed the first two or three words of a new line. After three months, on April 1, he could see a large object move quite

at the periphery on his left side, and he could see a small disc of white paper and count fingers at 45°.

In Migraine the onset of hemianopia is generally sudden. One instance of this will suffice:

N. H., a woman, about 35 years of age, has been liable to sick headaches since girlhood. When aged 23 she one day suddenly noticed that she could see only the half of anything she was looking at. Becoming alarmed, she ran to a looking-glass and then noticed she could see only the reflection of the right side of her face, as far as the middle of her forehead and nose. This condition lasted about five minutes, and was followed soon by severe headache and by vomiting several hours after.

The pathology of this visual affection is disputed. Galezowski, Du Bois Raymond, Latham, and others ascribing it to vaso-motor spasm, as evidenced by the change of colour of the face. Liveing puts forward the theory of a "nerve-storm," and Gowers (14c) says, "The hypothesis that the derangement is primarily one of nerve cells of the brain enables us better to understand the relation to other neuroses, and especially that to epilepsy, which is occasionally so distinct. . . . . These relations, however, make it intelligible that the two should occur in the same subject, and that intermediate forms of nerve disturbance should sometimes be met with."

Dr. Hughlings Jackson (38) speaks of migraine as a sensory epilepsy, the discharging lesion being in the posterior lobes, the ocular disorder and unilateral disorder of sensation as parts of the paroxysm, the headache and vomiting post-paroxysmal. He thinks these sensory epilepsies bear the same relation to hemianæsthesia with hemianopia from disease of the optic thalamus as unilaterally beginning convulsions do to the ordinary kind of hemiplegia from destruction of the corpus striatum.

Sir Samuel Wilks (41), on the other hand, sees no resemblance between migraine and epilepsy. He says they never pass one into the other, they do not occur in the same families or in the same class of persons, and each is relieved by different drugs.

Dr. Auld (36) considers migraine to be a functional

neurosis, and that the existence of a cerebral lesion is not established on satisfactory grounds.

Savage (40) considers that "migraine is undoubtedly a neurosis, occurring in families in which other forms of nervous instability are met with, and it is noteworthy that in some of these cases insanity develops, and that almost invariably with the onset of insanity the tendency to the recurrence of the headache ceases."

H. C. Wood (42), in a clinical lecture on "Epileptoid Migraine" at the University of Pennsylvania, showed a patient who suffered from typical epileptic attacks, with loss of consciousness and tongue biting, always preceded by a visual aura on his right side resembling the well-known fortification spectrum of migraine, which then faded, leaving a spot of darkness which increased until the whole field was obscured. Later, this patient suffered from minor attacks without loss of consciousness, preceded by a similar aura lasting twenty to forty minutes, and followed by violent temporal headache, nausea, and depression, in fact attacks indistinguishable from migraine. In spite of this, Dr. Wood has seen only two other cases in which there was any reason for suspecting the co-existence of epilepsy and migraine.

J. W. Gill (37) mentions two cases of recurring attacks of homonymous hemianopia, one occurring in a miner aged 20, who, after returning home, suddenly noticed he could see only the left half of any object. "At the same time an indescribable anxiety seized him, quite out of proportion to his concern at losing half his field of vision, and at the same time he felt a fulness at the epigastrium, as though he had wind on the stomach." After lasting a few minutes, a vibratory movement appeared in the blind field, gradually increasing, and then fading, until in about fifteen minutes his sight was again normal. Scarcely any headache followed the attack, and he has had similar attacks, at intervals varying from a few days to a few months, for the last forty-five years, though not so intense of late years, and varying in intensity and in duration from a few minutes to an hour. There was no history of epilepsy or of any nervous disease in the family.

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Another case had similar attacks at an interval of four years, and then recurring at intervals of a few days. Such cases are indeed strongly suggestive of a close relationship between epilepsy and migraine. Gowers (48) has "met with cases in which epilepsy succeeded migraine, and the epileptic fits seemed, as it were, to grow out of the attacks of migraine, being preceded by such sensory symptoms as had occurred before the attacks of headache. . . . In rare cases of epilepsy, again, a visual aura may consist of fortification-spectra with colours, and even, as I have known, with hemianopia. In one such case the visual disturbance lasted ten minutes, occurring sometimes alone, sometimes with transient loss of consciousness, sometimes with a convulsive attack." He goes on to say (49), "In most cases of epilepsy with a visual aura, this is brief, lasting only a few seconds, while the visual disturbance in migraine lasts twenty minutes to half an hour."

During the last eighteen months I have had the opportunity of studying four patients, who have had between them eleven attacks of transient hemianopia, each attack lasting for several hours, and generally accompanying unilateral convulsions, but in one case definitely preceding a typical epileptic fit. These cases form, I think, a further link in the relationship of epilepsy to migraine.

J. R., a butcher, while walking to his work one morning in September 1895, his eyesight suddenly failed, and when he reached the shop he could see scarcely anything. He became dizzy, was put on a sofa, but rolled off, losing consciousness for five hours. He thinks he had no convulsion, and on recovering consciousness his sight had returned. Since then his sight has failed similarly on several occasions, and he has had numbness and tingling in both arms and in the left leg during the attacks, but no twitching. A history of syphilis acquired twenty-five years ago was obtained.

On admission into the Queen Square Hospital in March, 1897, under Dr. Beevor's care, he seemed fairly well, no weakness in any limb nor any anæsthesia, gait and coordination normal. Vision $\frac{6}{12}$ in each eye, visual fields normal, ocular movements, pupils and discs normal. Knee jerks normal and equal.

On March 25, on getting up at 5.30 a.m. he felt dizzy, as

though he should be sick, and his sight seemed blurred. He went out for a walk about 11 a.m. with the other patients, and thinks his sight was gradually getting worse up to this time. While out he noticed numbness, first in the left hand and then in the right, spreading thence to the feet, with a tingling sensation running up from the left knee to the hip. He was observed not to notice objects or people on his left, and to bump up against them. When I saw him on his return at 12.15 p.m. he was complaining of feeling unwell, with pain across the forehead, and dimness of sight. On testing his fields of vision at once there was found to be complete left hemianopia for light, form, and colour, and with most careful testing, by the method above described, neither a small piece of white paper nor the flame of a match was seen by either eye on the left side until the fixation line was reached. Charts were at once taken with McHardy's perimeter, with the same result, showing scarcely any contraction of the remaining half-fields for white, though the colour fields were markedly constricted.

His pupils were normal, with no hemianopic pupil reaction, and they reacted well to light and accommodation.

The ocular movements were normal, except for slowness in turning the eyes to the left.

Special senses. No affection of smell or taste, but he could not hear a watch on the left side on contact, though on the right side he could hear it at four inches. Vision was as before, $\frac{6}{12}$ in each eye. No word blindness. Slight weakness of the left arm also was noticed, and anæsthesia to light touches of the left hand and fingers. There was no weakness of the leg, and his speech and articulation were normal. Intelligence and memory perfect. Knee jerks normal; no increase of either wrist jerk.

He was put to bed, and given an injection of 3 minims of liq. Strych. with a dose of half an ounce of brandy and a teaspoonful of sal volatile. Three hours later, at 3.30 p.m. he had a typical epileptic fit, with convulsion of both arms and of the face, and total loss of consciousness, lasting for five minutes, though afterwards he knew nothing of it. At 6 p.m. two-and-a-half hours after the fit, the left hemianopia was found still complete for all forms up to the fixation point, as before. The numbness, anæsthesia and weakness of the left hand had passed off, and he could then hear a watch two inches distant from his left ear. He still had pain across the forehead and back of head, worse on the right side, with some tenderness behind the right ear and on the right side of the occiput.

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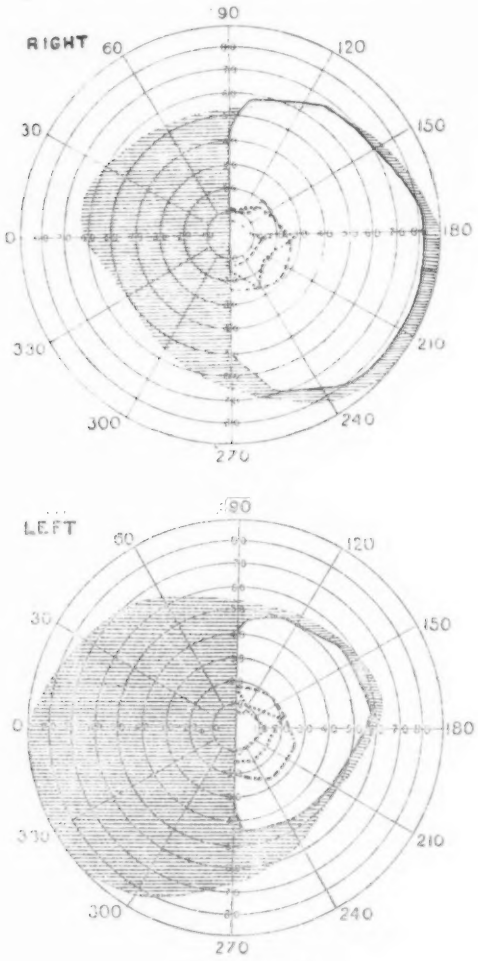

----- Blue field.
..... Red field.
-.-.-.- Green field.

FIG. 7.

J. R.—Case of transient hemianopia, with constriction of remaining colour fields. The dividing line cuts the fixation point.—*March 25th*, 1897.

Next morning at 9 a.m. the left hemianopia was clearing up. He could then see a large object, as a hand, moving at 80° on the left from the line of fixation, though he could not count fingers further out than 25°, nor recognise any colour until it nearly reached the fixation line on the left side, though his colour fields on the right side were again normal. A small white disc, 1 cm. in diameter, such as is generally used with the perimeter, he could see at 30° to his left. In the evening his visual fields had again become perfectly normal. The hemianopia was therefore absolute for at least twelve hours, and about the same time elapsed before it completely cleared up. Again, on April 5, at 7 a.m. his vision became impaired, and when seen at 12 noon, there was incomplete left hemianopia. He could see a hand moving at 80° to his left, and a small white disc at 45°, though he could not see the red head of a pin, nor recognise its red colour, until near the fixation line. The object, however, became bright and clear before the fixation line was reached, an evidence of the escape on this occasion of the centre for the macula in the cortex, or else of its earlier recovery. At 4.30 p.m. his left field was still much impaired, and he could not distinguish between one and two fingers held up further out than 20° to his left, though he himself, and most people I have tested, can ordinarily in a good light, distinguish between one and two fingers held up at 80° from the fixation line. Next morning his visual fields were again normal, no fit having followed the visual disturbance on this occasion. He was then ordered by Dr. Beevor 30 grains of pot. brom. every night, since when he has had no further attack.

He now says that the attacks he has had of dimness of sight during the last eighteen months have been of precisely similar character, though less severe, and occurring about once a month. Their resemblance to the hemianopia occurring in migraine, being followed by headache, is remarkable, though they persist much longer, and the definite association with epilepsy in this case points strongly to the supposition that in this transient hemianopia we have evidence of a primary sensory discharge in or near one half vision centre, generally proceeding no further, but occasionally overflowing and giving rise to an epileptic fit with loss of consciousness; the hemianopia thus appearing as an aura, though lasting very much longer than any previously de-

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scribed aura in epilepsy, and persisting for hours after the fit.

This long duration of the visual loss, especially in view of the result of the *post-mortem* in the case next to be described, is strongly suggestive of an organic lesion, hæmorrhage or softening, in the neighbourhood of the right cuneus, though the entire recovery of the visual field would preclude actual damage to the cuneus itself or the optic radiations.

A somewhat similar case, though of greater value since its completion by an autopsy, is that of a woman A. B., aged 53, who was admitted into the Queen Square Hospital in August, 1896, under the care of Dr. Hughlings Jackson. On her admission she presented the typical appearance of myxœdema, though eleven years previously, in 1885, she had been under Mr. Lawson's care at Moorfields for exophthalmic goitre, and an old photograph taken at that time shows the characteristic goitre and proptosis. These symptoms had persisted for about a year, when one night, ten years ago, she had a sort of fit, described as a "throbbing of the muscles," with partial loss of consciousness. Since that time she has had a similar fit about every three months, and for the last few months every month. Six months ago she had two severe fits with struggling. The history excluded the possibility of venereal disease. During the last two years her facial expression had been getting heavy, and slowness of speech had been noticed during the last twelve months. Three months ago, after a fit, she was delirious, and had aural and visual hallucinations. When seen in August, 1896, she appeared to be a typical case of myxœdema, with heavy expressionless features, malar flush, dry, scaly skin, hair coarse and dry, speech slow and drawling, with very impaired memory. Her weight was 9 st. 2 lb., though formerly it had been 14 st. The temperature was generally subnormal. No thyroid could be felt in the neck. She was weighed daily for a week, and then treatment was commenced by giving her thyroid tabloids by the mouth, at first one of Burroughs and Welcome's tabloids every other night, the dose being increased early in September to two tabloids daily. Her weight fell 3 lbs. at the end of the first week of treatment, and continued to fall until December 3, when she had lost 2 st. in weight. In the middle of December the dose of thyroid was reduced to half a tabloid every other night, which it was found was all she could bear, as when taking more she lost all appetite.

Briefly, she improved steadily under the treatment, and the symptoms of myxœdema gradually disappeared, and she became brighter and more cheerful, though her memory was always bad for recent events. When discharged on March 28th, 1897, she weighed 8 st. 4 lbs., being 12 lbs. less than on admission.

Between October, 1896 and January, 1897, she had five fits, all of them left sided, and very similar in character, so that a description of the first will apply fairly closely to the succeeding attacks.

On October 20, at 1 p.m., she was suddenly seized with left-sided convulsions, her head and eyes being turned to the left, with twitching of the left hand and foot. Consciousness was only partially lost, and she could turn her eyes to the right when loudly told to do so. The twitchings of the left hand and foot lasted for half an hour, and were followed by complete paralysis of the left arm, and weakness of the left leg. There was no hemianæsthesia nor any affection of taste, smell, or hearing, but there was complete left hemianopia, for light as well as for form and colour, the dividing line between the seeing and the blind halves, after the most careful testing, being found to pass exactly through the fixation point. The paralysis of the left hand was complete for five hours, but was being recovered from during the same evening. Next morning at 11.30 the left hemianopia was still complete, and charts were taken with McHardy's perimeter. She was otherwise fairly well, except for some headache. Her vision was $\frac{6}{24}$ in each eye, much the same as before the attack. Towards that evening the hemianopia began to pass off, vision improving from the centre towards the periphery. Two days after the attack there was scarcely any contraction of the left field remaining, and on the third day her visual fields were again perfectly normal.

As in the case previously related, there was absolute left hemianopia lasting for several hours, in her case for twenty-eight hours, and then vision gradually cleared up again to normal. In the case of the man, J. R., the visual disturbance was found a considerable time before the onset of the fit, and amounted to a definite aura, but though in the case of the woman A. B. the hemianopia was not found until attention was drawn to her case by the convulsion, yet in view of the much longer time that the hemianopia lasted as compared with the paralysis of the left arm and leg, it seems

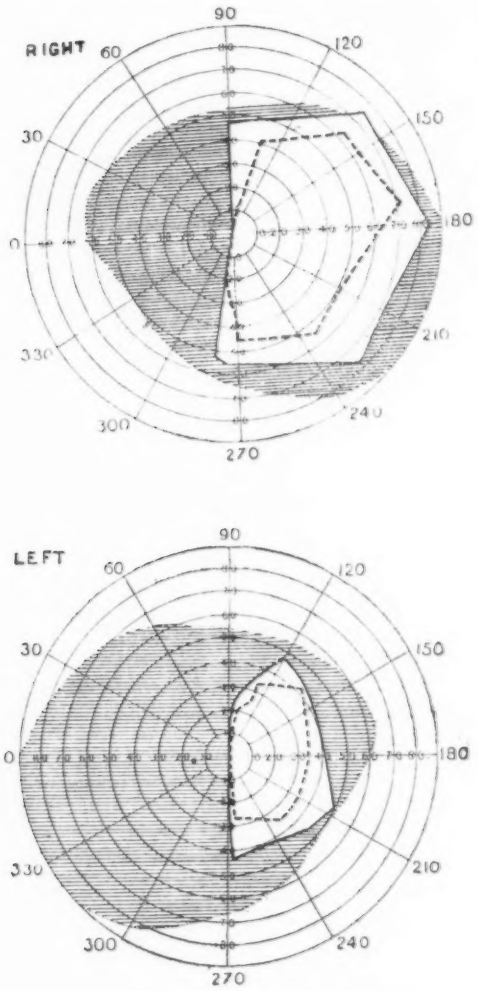

FIG. 8.

A B.—Case of left transient hemianopia. No contraction of remaining colour fields. The dividing line cuts the fixation point. *October 21st*, 1896.

reasonable to suppose that the primary discharge in her case also commenced in or near the half vision centre on the right side and spread thence to the Rolandic area.

She had similar fits on four other occasions while in hospital, on November 19, November 28, December 22, and on January 20. Both she and her friends also were quite sure that all the fits she has ever had have been of the same character. In each there was slight twitching of the left arm and leg, followed by temporary left-sided weakness and complete left hemianopia, which lasted for about twenty-four hours, and then cleared up again in two days to normal vision. In each instance the dividing line between the seeing and the blind halves was found by careful testing to cut the fixation point, and this observation was confirmed by Mr. Marcus Gunn, who saw her during the hemianopic stage on December 22. On two occasions there was also noticed after the fits temporary weakness of the lower part of the face on the right side, with partial aphasia and word blindness. She could never write anything, not even her own name, during all the time she was in hospital, though formerly she could write well. She was right-handed.

The charts of her visual fields, taken during the stages of hemianopia, showed scarcely any contraction of the remaining half fields, nor were the colour fields at all contracted as in the case of J. R. After the fit on January 20, Dr. Taylor ordered her 30 grs. of bromide of potassium every night, and she never had another fit up to the time of her death on April 20. She was discharged on March 28, and continued taking the bromide every night, and half a thyroid tabloid every other night while she was at home. She soon contracted influenza and pneumonia, and died three weeks after leaving hospital.

Autopsy.—April 23, sixty-seven hours after death. An examination of the head only was allowed. The brain, though soft in places, appeared perfectly normal. There was no thickening of the membranes, and the pia mater stripped readily. Close examination of the cuneus and of the motor convolutions on each side revealed nothing abnormal, nor was any softening seen on section of either cuneus. The brain was then hardened in Müller's fluid, and on dissection two months later, a small cyst was found in the lower part of the right quadrate lobule. The cyst

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was the size of a very small marble, about a quarter of an inch in diameter, and was evidently the result of an old hæmorrhage, containing granular yellowish pigment. It was placed just in front of the parieto-occipital fissure, in the white matter at the depth of about one third of an inch from the surface, on the internal aspect of the quadrate lobule. Microscopical sections of the surrounding brain substance showed no organised cyst wall, but sections stained by Marchi's method showed numerous degenerated white fibres. The nerve cells appeared quite normal, and no other lesion could be found in the brain.

The origin of her attacks is thus explained. In all probability her first fit which occurred ten years ago, when her symptoms of Graves' disease were well marked, was due to the occurrence of the hæmorrhage, and the recurrence at varying intervals since, of left-sided convulsion were due to instability of surrounding grey matter, set up by the irritation of the softened and necrosed white matter beneath.

The situation of the lesion, in such close proximity to the right cuneus and optic radiations, will account for the absolute left hemianopia which was found always accompanying the fits, and which was, no doubt, one of the initial occurrences, probably preceding the motor convulsion, and due to early loss of function of the half-vision centre.

Comparing then her case with that of the man previously related, we have an illustration of two different kinds of fit, each started by a "discharge of nerve force," whether originating in the nerve cells themselves or in the interfibrillary network beyond them, in or near the half vision centre, or right cuneus. In the man's case, a general convulsion with early total loss of consciousness, that is to say, an ordinary epileptic fit resulted, while in the other the discharge was more localised to one side of the brain, affecting the motor convolutions chiefly, and giving rise to unilateral convulsion with only partial loss of consciousness, or a Jacksonian fit. It is well known that aphasia, especially word deafness, may occur as a temporary symptom in migraine, and though the occurrence of a convulsion is said to exclude migraine, yet I think the attacks in this second

case bear some similarity both to migraine and to ordinary epilepsy. It is, perhaps, a significant fact that a daughter of this patient, aged 18, suffers from typical migraine with right sided visual spectra, though, so far as I am aware, without hemianopia.

Another case which closely resembles the last is that of a woman, E. B., aged 40, who was admitted into the Queen Square Hospital, under Dr. Bastian, in March, 1896. She had a history of headache, and of numbness of the left hand for five years, and during the last eighteen months she had three left-sided convulsive attacks. There was no history of syphilis. On admission she had slight left hemiplegia, chiefly affecting the arm, with marked wasting of the intrinsic muscles of the left hand. Her temperament was nervous and excitable. On April 1, at 7.30 a.m. she had a fit, preceded for half-an-hour by numbness in the left fingers, excitability and continual talking, with a flushed face and widely open eyes. Her left hand, left arm and leg then began to shake, and she felt a sensation of twitching in the left side of the face, though none was observed. The twitching persisted longest in the leg, and was followed, one and-a-half hours from the commencement of the attack, by a general convulsion with loss of consciousness, cyanosis, and turning of the eyes to the right. When I saw her at 11 a.m. she could move her left shoulder and elbow, but not the wrist or fingers, and there was distinct loss of sense of position in the left fingers, wrist and elbow. There was partial left hemianæsthesia to touch and pin prick, scarcely more marked on the hand than elsewhere. There was also complete left hemianopia, reaching close up to the mid line, though, this being the first case of the kind I had seen, I did not specially test the relation of the dividing line to the fixation point, beyond taking perimeter charts. There was no affection of either smell or taste, but her hearing in the left ear was much diminished, being $\frac{1}{40}$, with diminished bone conduction. Her pupils were equal and normal, and vision was R. V. $= \frac{6}{60}$ J6, L. V. $= \frac{6}{12}$ J1 nearly. There was no weakness of the left leg, but the left knee jerk was much increased, with left ankle clonus. Four hours later she had recovered a good deal of power in the left fingers and wrist, the left hemianæsthesia had nearly disappeared, and the sense of position in the fingers and hand had improved. Her hearing had then improved to $\frac{6}{40}$ on the left side, and the left hemianopia was also clearing up. When a black pen was moved inwards from the periphery she could tell

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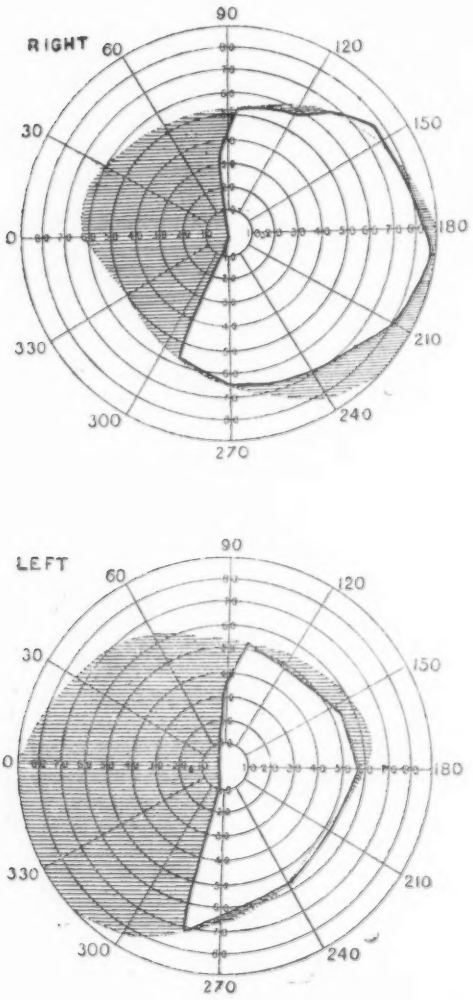

FIG. 9.

E. B.—Case of transient hemianopia.

something was moving at 60° from the centre, though she could not recognise the object until it was within 30°, and she could not distinguish between one and two fingers held up at 45°. By the next morning her visual fields had entirely recovered. Her hearing was $\frac{40}{40}$ each side, and there was no weakness or hemianæsthesia left.

On April 29, after complaining of headache the previous night, she had an attack at 7 a.m., commencing by numbness in the two ulnar fingers of the left hand, spreading up the arm to the shoulder which then commenced to shake, followed by flexion of the forearm and twitching of the fingers of the left hand for a few minutes. There was no movement of the head or eyes, no general convulsion, nor any loss of consciousness. An hour later she had a left sided visual spectrum, commencing as a bright point on her left side. This rapidly enlarged to a ring, the size of the palm of a hand seen at a distance of three feet, being coloured brilliantly in green and gold, and pulsating synchronously with her heart beat, which she could feel as a throbbing sensation all over her body. This was followed by visual hallucinations of people in wheel chairs, wheeling them from left to right as far as the mid line, all brilliantly coloured also in green and gold. On this occasion there was no hemianopia or contraction of the field, no hemianæsthesia or affection of any special sense, except hearing, which was again diminished temporarily on the left side to $\frac{5}{40}$. The visual spectra lasted for about half-an-hour, and did not recur again whilst she was in the hospital, though she says she has seen occasionally coloured curtains appear to come down in front of her, always to the left.

In this case the left hemianopia found after the first fit persisted for a much shorter time than in any of my other cases, being complete for only four or five hours, and then clearing up gradually *pari passu* with the disappearance of the left sided deafness, weakness, and hemianæsthesia. This fact, in view of the definite symptoms pointing to a lesion of the motor tract on the right side, probably in the cortical centre for the arm, make it probable that the primary focus of discharge occurred in the arm or hand centre and spread thence to the half vision centre on the same side, paralyzing its functions, and producing left hemianopia in the same way as the temporary weakness of the left arm and hemianæsthesia followed the left sided convulsion.

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That this was the real sequence of events is made more probable by the nature of the second attack, nearly a month later, in which numbness of the left hand and arm was followed by convulsion of the left arm, and, after another hour's interval, by a left sided visual spectrum and hallucinations. Her description of this spectrum closely resembles that so frequently found in migraine, though the sequence of definite hallucinations of moving figures in the left field, all brilliantly coloured in green and gold, is, I think, unique. This visual spectrum and the hallucinations, occurring without hemianopia, would probably indicate that the higher centre in the angular gyrus was also affected by the progress of the epileptiform discharge, rather than the half-vision centre only. This case again forms a curious link in the resemblance and relationship of migraine, Jacksonian fits, and ordinary epilepsy. Jacksonian convulsions, however, like ordinary epileptic fits, are but rarely accompanied by hemianopia, and in several other cases of hemiplegia accompanied by recurring unilateral convulsions, sometimes so severe as to produce total loss of consciousness, and also in post-hemiplegic epilepsy, I have never found any affection of the fields of vision, even immediately after the fits. It is probable, therefore, that the half vision centre is not liable to become paralyzed by even a severe discharge taking place in neighbouring centres unless (1) it is already slightly damaged, or (2) if hypersensitive and prone to spontaneous discharge as in migraine.

The temporary unilateral deafness which followed both attacks in this case, and also in the case of J. R. previously related, may be due to similar spread of nerve discharge temporarily paralyzing the auditory centres.

In two other cases I have seen there has been permanent partial hemianopia, due probably to cortical lesions, in each case associated with recurring convulsions and temporary complete blindness of the half fields on one side.

W. B., aged 54, a gas engineer, was admitted into the Queen Square Hospital under Dr. Beevor on December 30, 1896, suffering from partial right hemianopia, word blindness and agraphia. During the last eight months he has had a fit every

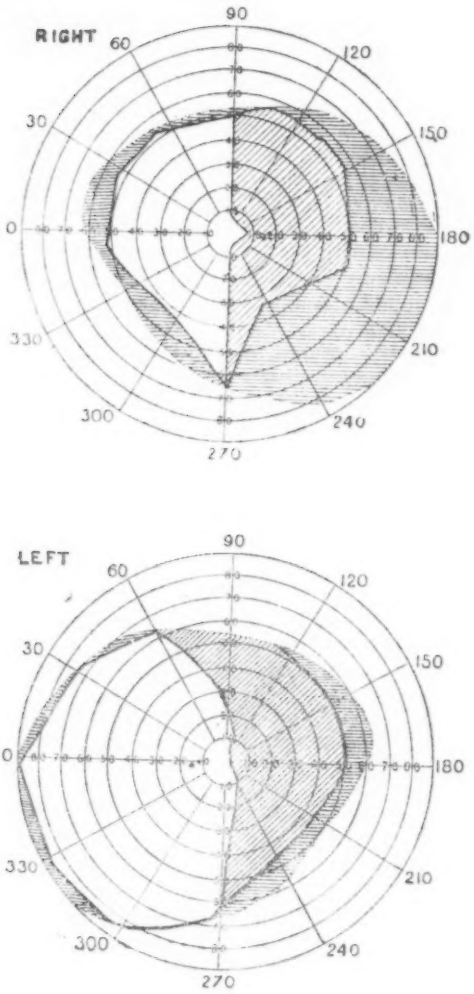

FIG. 10

W. B.—Case of permanent partial right hemianopia. The area of indistinct vision is indicated by lighter shading. Fields for white spot 1 cm. in diameter.—*January 3rd*, 1897.

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month, the last occurring a fortnight before his admission. He says he loses consciousness early in the fit and his friends say he is "drawn" on the right side during the attack, and afterwards his right arm and leg are weak for a time. He knows when the fits are coming on by a sensation of an electric feeling rising from his feet and spreading all over him. He has had frequent visual hallucinations of faces he knows, especially of his children, animals, &c., always on his right side, sometimes before the commencement of a fit. He denies even exposure to syphilis. On admission his heart was very irregular in action, though there was no murmur to be heard, and he has never had rheumatism. There was no weakness of either arm or leg, the knee-jerks were slightly increased and equal, and his gait natural. He was quite sensible, though his manner was generally a little odd and exaggerated, possibly owing to his difficulty in expressing himself. He had great difficulty in reading, though he understood all that was said to him, and he could pick out any object asked for at once, though he had some difficulty in naming them, but he could write scarcely anything. The eye movements, pupils and discs were normal, and his vision R. V. $= \frac{6}{18}$, L. V. $= \frac{6}{9}$. There was incomplete right hemianopia, the inner part of each right field appearing blurred, and an object not seeming distinct until near the fixation point. The outer part of each right field beyond 50° was completely blind. Colours were recognised on the left side from 20°—30° from the centre.

His vision improved during the first three weeks, so that he could tell when a hand or a large object moved even quite at the periphery on the right side, and he could count fingers at 45°. On January 24, he had two severe epileptic fits, with half-an-hour's interval between them. For some hours previously he had felt dizzy and unwell and was more incoherent than usual. At 3.30 p.m. the fit commenced, and Dr. Stewart's account is that it began suddenly with turning of the head and eyes to the right; he then became completely rigid, and the right side of his face twitched, followed by general convulsion. There was early and complete loss of consciousness, with absence of the corneal reflex, and dilated insensitive pupils. Slight double ankle clonus was present. The fit lasted two minutes, and half-an-hour later he had another precisely similar fit. When I saw him the next morning, there was complete right hemianopia up to the middle line, except for a bright light which was perceived nearly out to the periphery. He could not see any object or a

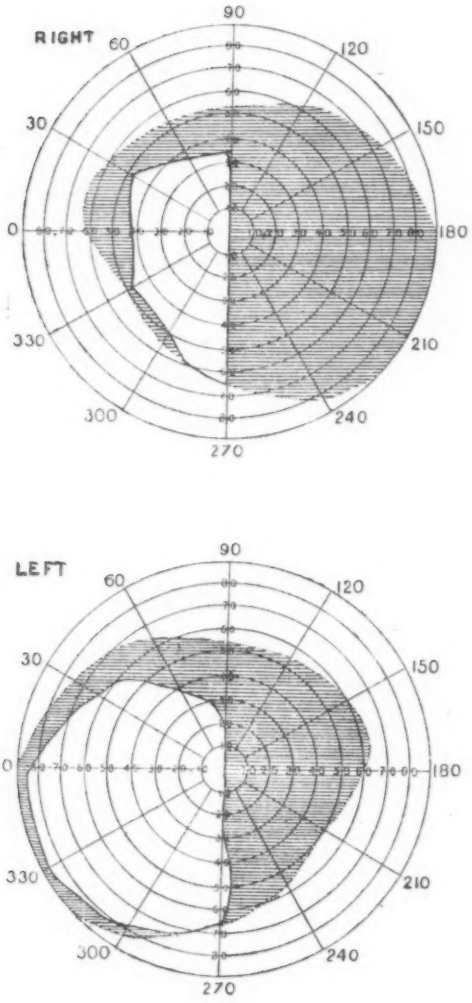

FIG. 11.

W. B.—Case of transient right hemianopia, the dividing line cutting the fixation point.—*January 25th*, 1897.

piece of white paper move until it exactly crossed the line of fixation, which observation Dr. Beevor confirmed at two o'clock in the afternoon. In addition, he was completely word blind, and could not name nor pick out objects, except coins, though he understood nearly all that was said. Late in the afternoon the hemianopia began to clear up slightly, vision returning first at the centre. The next day he could see a large object move at 30° from the centre on the right side, but he could not see a small piece of white paper until near the mid line. The visual field gradually enlarged again, and on the fifth day after the attack he could see a hand move at the periphery on the right side, and could count fingers at 45°, and could recognise colours at 30°. On February 20, he had an attack of localized convulsions on the right side, commencing in the thumb and finger, and spreading to the elbow and shoulder. There was no turning of the head or eyes, nor any loss of consciousness. The convulsions lasted for two hours, and, on testing him while they continued, there was found complete right hemianopia, this time for light as well as for form and colour, the dividing line cutting the fixation point. This remained complete until the next day, when it began to clear up gradually as before. The convulsions were followed by some weakness of the right hand and arm, which had disappeared on the following day, considerably before the recovery of the visual field. Just a month later, on March 23, he had another similar attack of localized convulsions on the right side without loss of consciousness. In the afternoon he complained of feeling unwell, and went back to bed. I saw him at once, and noticed slight twitchings of the right thumb and fingers. On testing the visual field immediately, there was found again absolute right hemianopia, the dividing line in each eye cutting the fixation point, as before. His right hand and arm were very weak, and he could not grip at all, though the leg was as strong as usual. He was almost entirely aphasic, and could not name anything, nor read a word, though he could ejaculate a few sentences; and he was also partially word deaf. He recognised, however, the difference in value between coins. The twitchings of the hand became more violent, and after continuing for nearly two hours were stopped by a dose of 25 grs. of chloral hydrate and 20 grs. of bromide of potassium. On the next day at noon he was still completely hemianopic on the right side, but the aphasia and word blindness had improved considerably. By the following morning, the right visual field had again much improved, and he could see a hand move at the periphery. Dr. Beevor then

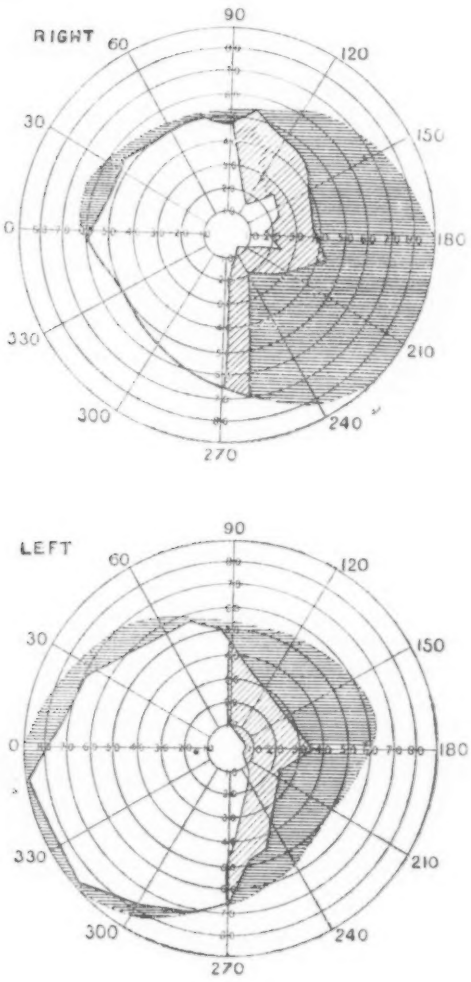

FIG. 12.

W. B.—Partial right hemianopia, persisting after an attack of transient complete hemianopia.—*February 2nd*, 1897.

Fields for white spot 1 cm. in diameter the area of indistinct vision being indicated by lighter shading.

ordered him 30 grs. of bromide of potassium every night, and since then up to the time of writing (June 1) he had not had another fit.

While in hospital he thus had three separate attacks, which on the first occasion resembled ordinary epileptic fits, the two latter being typical Jacksonian convulsions without any loss of consciousness. Each attack was, however, accompanied by complete right hemianopia, which was found during the last attack to be present as soon as slight twitching of the hand had begun, though he was not tested on the first occasion until eighteen hours after the fit, and on the second occasion not until two hours after the commencement of the convulsions. Moreover, the patient is quite certain that he has had similar disturbance of vision accompanying each of the fits he has had at home.

In view therefore, of (1) the probable early occurrence of the hemianopia in all the attacks (2) its long duration, being complete for about twenty-four hours, and then the visual field gradually improving again to its previous condition, and (3) the partial persistent impairment of the right half fields, which is evidence of some lesion in the left half vision centre or visual fibres, it seems probable that in this case again we have an instance of a primary epileptic discharge near the left cuneus, temporarily but completely abolishing its function. In his case this led on some occasions to total loss of consciousness with general convulsion, or an ordinary epileptic fit, while on two occasions Jacksonian fits have followed, an illustration, I think, in the same patient, of the possible variety in the spread of an epileptic discharge originating from the same focus.

An interesting feature of the case was the complete word blindness, and slight word deafness found after the attacks, which, taken with the persistent partial word blindness and agraphia, indicates some lesion of the visual word centre in the left angular gyrus. This might be a separate patch of softening, or what is perhaps more probable, the angular gyrus may be partially damaged by softening, which extends deeply into the white matter, and so has damaged the optic radiations which run in close proximity, thus causing the

partial persistent hemianopia. The primary epileptic discharge may then be assumed to take place in the higher visual centre, and, spreading thence cause the temporary complete hemianopia by paralyzing the half-vision centre on the same side. Word-deafness and word-blindness in association with *petit mal* have been recorded by Dr. Hughlings Jackson (46).

A history resembling those of the previous cases, I met with recently in an out-patient of Dr. Luff's, whom I had the opportunity of examining at St. Mary's Hospital.

A woman, E. W., aged 51, while complaining of some gastric trouble, mentioned that she had numerous attacks beginning with "dazzling in the eyes" on the left side, followed by a partial loss of sight, and by tingling in the left arm and leg, and convulsive twitchings of the left hand and forearm. She then used to feel a curious sensation of swelling in her stomach followed by loss of consciousness.

Though I have not seen her in an attack I have little doubt that her case would prove to be a similar one to those previously related, and that the partial loss of sight, if investigated at the time of an attack, would be found to be left hemianopia.

Two other cases I have seen in which transient hemianopia was associated with unilateral convulsions, and also with severe epileptic fits, in one typical case of general paralysis, and in another obscure case which presented some symptoms also resembling general paralysis.

This latter case, J. H., a man, aged 36, had been discharged from the army in April, 1896, on account of his eyesight and inability to do his work. He had been in the army eighteen years, and had contracted syphilis when he first joined. The sight of his left eye had always been weak. He was fairly well until the end of the second week in November when his left hand and arm became weak, and he noticed that he bumped up against people on his left side. On admission to the Queen Square Hospital under Dr. Ferrier, on November 18, he was found to have marked weakness of the lower half of the left side of his face, and also of his left arm, though less marked. He could

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walk fairly well, and there was no weakness of either leg. There was marked tactile anæsthesia and loss of sense of position in the left hand, wrist and forearm; and also complete left hemianopia, and slight left hemianæsthesia, but the exact position of the dividing line could not be ascertained owing to his drowsiness and insufficient answers. His vision was R. V. $\frac{6}{9}$, L. V. $\frac{6}{18}$. There was compound hypermetropic astigmatism in each eye quite sufficient to account for the lowered acuity of vision. The optic discs were normal. There was also marked deafness of the left ear, with diminished bone conduction. Knee-jerks increased, L > R. Pupils unequal, L > R, neither reacting to light, though they did so to accommodation. The ocular movements were normal. He had a fit two days after admission, commencing with clonic contractions of the masseters, and movements of the tongue, palate and lips. There was no loss of consciousness, and he could answer questions while the convulsions were going on, which lasted about two minutes. He had numerous fits, closely resembling this one, during his first week in hospital, and then, during two days, he had three batches of strong epileptic fits with early total loss of consciousness and general convulsion, with cyanosis. He was placed on large doses of mercury and iodide of potassium, and had no more fits after the first ten days in hospital. He slowly began to improve, his mental condition became brighter and his left arm and face became again as strong as the right side. At the same time the left hemianopia began to improve gradually, and by the time of his discharge in April, he could tell when a hand was moved quite at the periphery on the left side, and he could count fingers at 45°. As he improved he began to show symptoms suggestive of general paralysis, the pupils remained unequal and inactive to light, his articulation was defective, with a tendency to clip the ends of words, and there was characteristic tremor of the tongue and facial muscles, with overaction of the frontalis and corrugator supercilii on showing the teeth. His manner too became slightly exalted, and he declared he was never better in his life.

The case curiously resembled that of a man, G. S., under the care of Sir William Gowers in the autumn of 1895, an account of which I published in the *Lancet* (23). In each case there was weakness of the left hand and arm, with slight left hemianæsthesia and left hemianopia. Both had localised convulsions affecting the left hand and the tongue, palate and face, and both had also batches of strong epileptic

fits with complete loss of consciousness. Both, moreover, bore a certain resemblance to cases of general paralysis.

In another case of a man, H. S., aged 40, a typical case of advanced general paralysis, I had the opportunity of watching two attacks of localized convulsions on the right side, each being followed by complete right hemianopia lasting for about ten minutes, though there was no hemianæsthesia and only slight motor weakness. He could not answer questions, but there was no palpebral reflex to a finger darted at his eyes from the right side, and he gave no sign of seeing any object approaching him on his right side until it crossed the middle line. In about ten minutes he began to perceive objects again to his right. Dr. Stoddart at Bethlem Hospital, and Dr. Cole of Moorcroft Asylum, have also informed me that they have found hemianopia, each on one occasion, in a case of general paralysis after an attack of unilateral convulsions, the hemianopia being on the same side as the convulsions, and disappearing completely after a short time.

I cannot help feeling, therefore, that if more cases of general paralysis were examined for this point after the fits, hemianopia would not infrequently be found, lasting for a longer or shorter interval. In general paralysis the destruction of the cortex is widespread, and the occurrence of transient hemianopia after the fits would be accounted for by the process beginning to affect the half vision centre, the primary explosion of nerve force originating the fit, whether occurring in the cuneus or spreading to it from the motor area, causing temporary exhaustion of its function. In Jacksonian epilepsy, or localized convulsions due to a limited lesion in the Rolandic area, I should not expect to find transient hemianopia, though I think it would be advisable to test all cases for it. I have not had the opportunity of testing this point in any cases of localized convulsions due to uræmia, in which condition I should think it not unlikely to occur. Amaurosis is well known as a transitory symptom, sometimes associated with uræmic convulsions, and it seems to me quite possible that some cases, if tested, might be found to develop transient hemianopia.

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Conclusions.

(1) That hemianopia, rarely binasal, more commonly lateral and left sided, with accompanying constriction of the remaining half fields, may occur as a temporary phenomenon in hysteria.

(2) That hemianopia due to a vascular lesion of the cuneus, of sudden onset, may commence with marked loss of sight, sometimes amounting to complete amaurosis, and due probably to inhibition of the remaining half vision centre.

(3) That the cortical half vision centres are not subdivided into centres for light, form, and colour respectively, and that hemiachromatopia may be due to a lesion anywhere in the visual path between the chiasma and the cortex.

(4) That quadrantic hemianopia, though strongly suggestive of a cortical lesion, may sometimes be due to a lesion in the internal capsule.

(5) That the macular region of the retina is invariably supplied with nerve fibres on the same plan as the rest of the retina, *i.e.*, each side of it from the corresponding side of the brain. That in all cases of absolute transient hemianopia the dividing line between the seeing and the blind halves invariably passes through the fixation point.

(6) That the cortical centre for the macular region in each cuneus is less liable to complete destruction and recovers earlier than the rest of the half vision centre.

(7) That cases of persistent hemianopia in which the dividing line passes to one side of the fixation point, leaving it in the seeing half, are to be accounted for, either (*a*) by the escape or partial recovery of the cortical centre for the macula, or, (*b*) by the acquirement by education of a new fixation point in the retina.

(8) That hemianopic visual spectra of low elaboration, such as red and green lights, or the varieties of scintillating scotoma in migraine, are caused by a discharge in the half vision centre in the cuneus.

(9) That complex visual phenomena of hemianopic type,

such as faces, animals, &c., are elaborated in a still higher visual centre, which possibly is the angular gyrus; their occurrence in the half field only being due to reflex irritation from a lesion generally in or near the cuneus, but which may be in the optic radiations or optic tract.

(10) That double hemianopia does not necessarily cause permanent amaurosis, in many cases the return of a small area of central vision indicating the escape or recovery of the cortical centre for the macula in the cuneus on each side.

(11) That the hemianopia in migraine is due to an epileptic discharge in the half vision centre of one side.

(12) That in many cases an epileptic discharge may originate in, or near, the half vision centre on one side, in some cases proceeding no further, beyond producing temporary hemianopia, in others producing a typical epileptic fit, and again in others giving rise to unilateral convulsions without loss of consciousness.

(13) That transient hemianopia in such attacks may last for twenty-four hours or longer, and may be due to vascular softening adjacent to, but not involving the visual centre or path.

(14) That transient hemianopia is rare in ordinary Jacksonian epilepsy, and is not liable to occur unless the half vision centre be (1) already slightly damaged, or (2) hypersensitive and prone to spontaneous discharge, as in migraine.

(15) That such transient hemianopia not unfrequently accompanies unilateral convulsions in general paralysis, and may possibly occur in uræmia.

(16) That the auditory centre may be similarly paralyzed through spread of the epileptic discharge.

Henschen (14a) has recently published a case of quadrantic hemianopia, the lower quadrants of the field being blind, due to a hæmorrhagic cyst in the posterior part of the pulvinar, which had destroyed the dorsal half of the external geniculate body, but left intact the optic tract and optic radiations, and he infers that the dorsal half of the external geniculate body corresponds to the upper quadrants of the retinæ, just as, in his opinion, the upper lip of

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the calcarine fissure corresponds to the same quadrants of the retinæ.

[Since writing the above, the patient, W. B., died suddenly on November 29 of cerebral hæmorrhage. At the autopsy there was found, in addition, a patch of softening in the second temporo-sphenoidal convolution on the left side, about two inches in length, and not quite reaching up to the angular gyrus. The cuneus on each side appears normal, but the brain has not yet been cut, and it is possible that the softening in the subjacent white matter may extend backwards beneath the angular gyrus.]

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NOTE ON MUSCLE-SPINDLES IN PSEUDO-HYPERTROPHIC PARALYSIS.

BY ALBERT S. GRÜNBAUM, M.A., M.D., M.R.C.P.

In 1894[1] Sherrington gave the first account of the structure and function of muscle-spindles based upon physiological experiment. A stimulus was thus given to their more thorough examination in pathological conditions.

Recently Batten has given the results obtained by him in the examination of cases of several nervous diseases, finding, however, except perhaps in tabes, no appreciable changes in the muscle-spindles. So far as I am aware, no account has yet been published of their condition in pseudo-hypertrophic paralysis, and in this disease I have been able to find some changes.

The material was obtained six hours after death, on a cold winter's day, and carefully preserved, but unfortunately, for the most part not histologically examined until over two years later.

In the vascular system there was considerable thickening of the smaller arteries, due, in part, apparently, to hypertrophy of the muscular layer.

In the central nervous system there was marked dilation of the perivascular lymphatics, and also of the blood-vessels. The latter often contained a large number of leucocytes. In the spinal cord were several small hæmorrhages.

In the peripheral nervous system there was degeneration of some fibres of the smaller nerves.

[1] In the last number of BRAIN, both in his historical *résumé* and in the bibliography, Batten erroneously gives the date of Sherrington's first paper as 1895. It should be 1894.

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In the muscular system as a whole the changes were of the ordinary character. In the gastro-cnemius very few muscle-fibres remained; the rest were entirely replaced by fat. In other muscles many fibres were atrophied, but some were distinctly hypertrophied. Most of them showed signs of alteration in the surrounding connective tissue, varying from a proliferation of nuclei of the sarcolemma to a deposit of fibrous tissue around the fibre. Several fibres had a distinctly hyaline appearance, and a few showed vacuolation.

The muscle-spindles were for the most part unaffected, but in a few there was a diminution in size of an intrafusal fibre with a deposit of hyaline material around. The nerve fibres supplying them were apparently healthy. In some muscles, *e.g.* gastrocnemius, I could not find any spindles in the sections examined.

Sherrington has shown that division of the nerve supplying a muscle will not produce atrophy or degeneration of the muscle fibres within the spindle. The muscle itself may be totally atrophied, and every nerve fibre going to it have disappeared, yet the muscle fibres within the spindle remain intact; the muscle-spindles are all that are left of the original muscle.

It would therefore appear highly probable that any pathological alteration in such fibres is primary, and consequently also primary in the other ordinary fibres, although commencing much earlier in them. So that, so far as it goes, my observation supports the now generally accepted theory of the intra-muscular origin of the disease.

This theory is also supported by the facts that generally no nervous changes have been detected in the cases examined, and the connective tissue changes do not appear to cause atrophy by compression, for where there is much connective tissue there is a total increase in the size of the muscle. It must be remembered, however, that the nervous and vascular changes, considered to be the primary cause by Erb and Babes respectively, were also present in my case.

I am indebted to Dr. T. D. Acland, under whose care the case was in the hospital, for permission to publish it.

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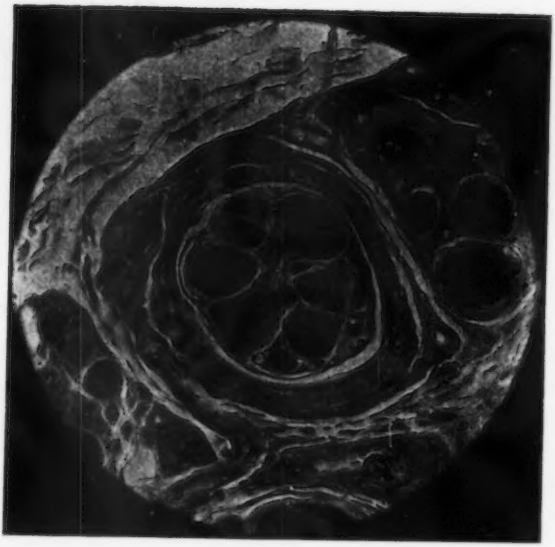

FIG 1.
Muscle-spindle in unaffected portion.

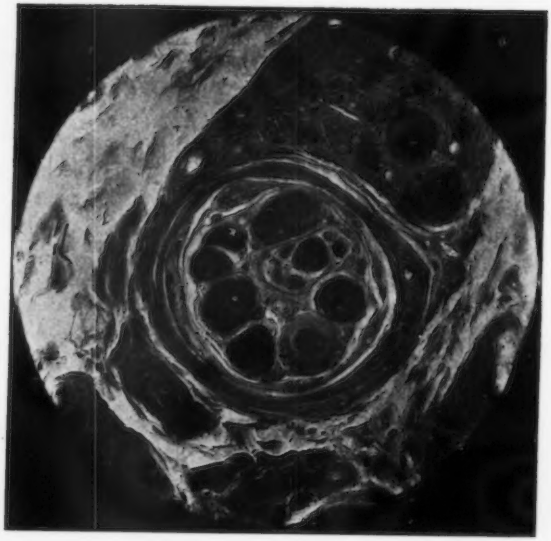

FIG. 2.
The same with affected fibre.

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FIG. 3.
Peripheral nerve, partly degenerated.

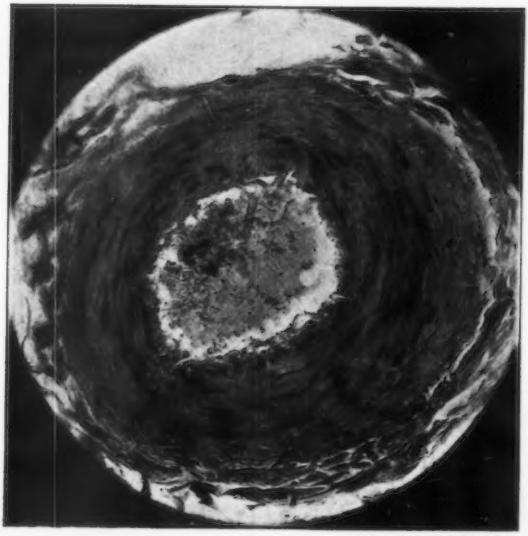

FIG. 4.
Artery with thickened muscular and external layers.

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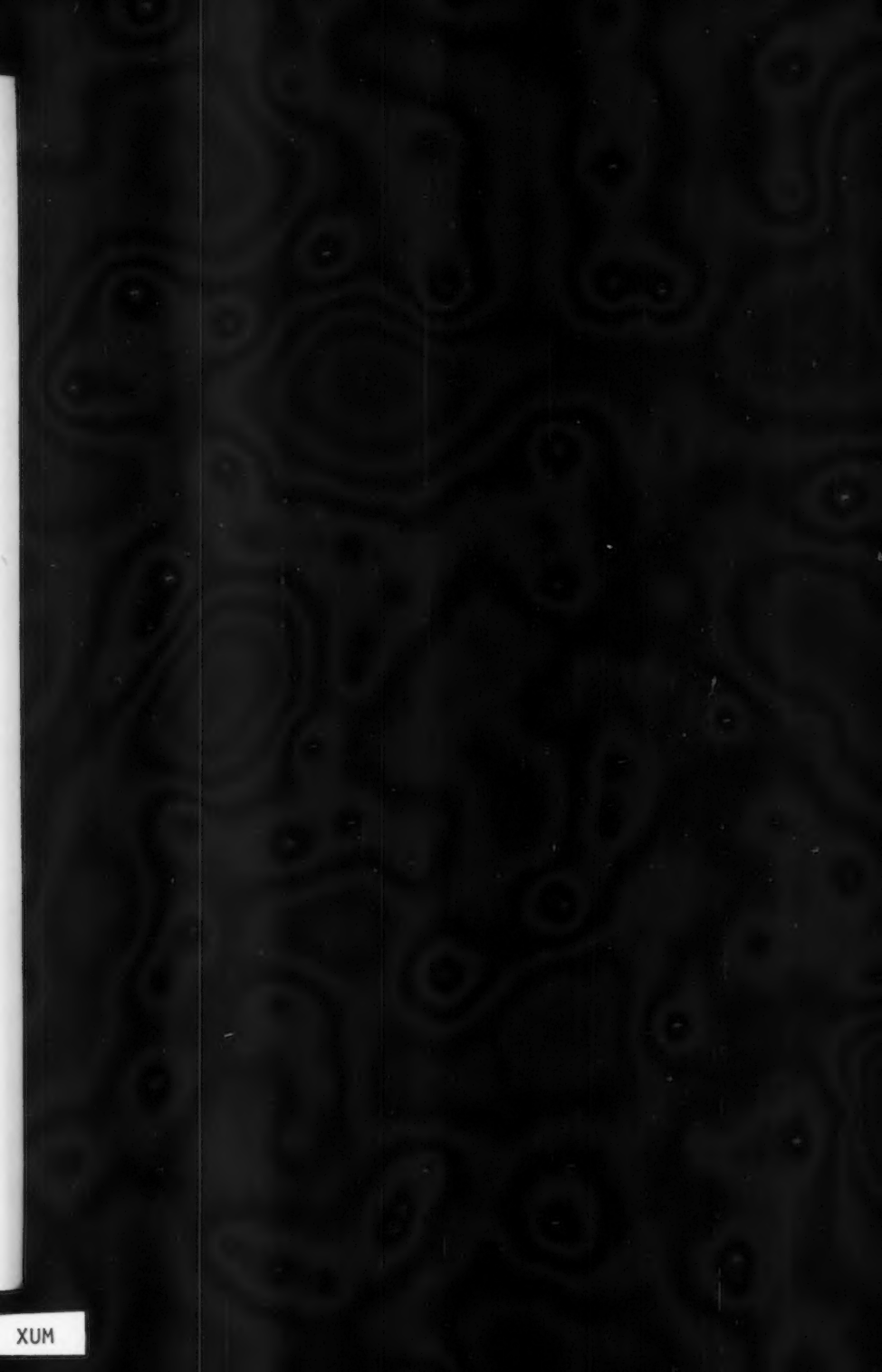
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Addendum.

After returning the proof of this article, a paper by Spiller[1] has come into my hands, from which it appears that he has already examined a case of muscular dystrophy (apparently not pseudo-hypertrophic paralysis). Spiller did not succeed in finding any change in the muscle-spindles.

References.

Batten. Brain, Spring and Summer, 1897, p. 138.
Sherrington. *Journ. of Physiology*, vol. xvii., 1894, p. 237.
Erb. *Samml. Klin. Vorträge.* Inn. Medic. No. 1, p. 1.

[1] Spiller, "The Neuro-muscular Bundles, &c.," *Journ. of Nervous and Mental Dis.*, Oct., 1897.

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OBSERVATIONS ON SENSORY NERVE-ENDINGS IN VOLUNTARY MUSCLES.

BY ANGELO RUFFINI.

Lecturer on Microscopical Anatomy, Royal University of Bologna.

THERE are at least three kinds of sensory end organs in voluntary muscles. Of these the most important is the muscle-spindle, and next to it comes the tendon organ; besides these the muscles possess a certain number of Pacinian corpuscles.

I.—MUSCLE-SPINDLES.

From the histological and physiological facts which we now possess regarding muscle-spindles, there can, I think, no longer be a question as to their nature being sensorial. It was already, in 1888, conjectured by Kerschner, but Kölliker in the following year rejected his arguments. In 1892 I myself was able to strengthen the view by contributing for the first time an accurate histological description of the nerve-ending; and in 1893 Sherrington actually demonstrated the existence of sensory nerves in muscle, and in the following year, by indisputable data obtained by experiment and microscopy, finally furnished conclusive proof that the spindle is an organ of sense.

In the past two years I have had the opportunity of preparing a large series of specimens of muscle-spindles from the cat. These have yielded me interesting results, which I will here summarise. In the muscle-spindles of the cat there exist three distinct types of nerve-endings, which I will call respectively the primary, secondary, and *plate* endings.

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The Primary Form of Ending.—This is the nerve-ending which I described minutely and figured in 1892, and Sherrington has confirmed my description of it. The large nerve-fibre which goes to form this ending just before it joins the spindle, almost always divides into two or more secondary branches; these branches, after having passed through the capsule of the spindle, divide into tertiary branches, each of which approaches a muscle-fibre in the intrafusal muscle-bundle, and terminates on it. The myelin sheath is lost on the tertiary branches, and each then becomes broad and flat and riband-like, and then either winds spirally round the muscle fibre or runs along one side of it as a longitudinal band from which, from point to point and at varying intervals, troop-like terminal expansions clasp the entire circumference of the fibre. Both these two modifications may co-exist and even lie juxtaposed in one and the same fibre. That is why it seemed, and still seems, to be well to give that ending the name of *annulo-spiral ribbon ending*. At the two extremities of this annulo-spiral ribbon ending the nerve terminates in diverse swollen knobs.

The Secondary Form of Ending.—Besides the primary there is found another form of ending distinct from it in morphological character and in the individuality of the nerve-fibre whence it springs. It has not yet been described in any paper, but I have known of it for some years. The parent nerve-fibre here also divides into secondary branches, but usually only after having penetrated the spindle. The secondary branches soon break up into a number of varicose axis-cylinders, united by very delicate and short filaments. The varicosity of the nerve-fibrils is of various kind, rounded, bifid, triangular, leaflet-like, &c.; the varicosities seem always disposed in such a way as to recall the arrangement of a festoon-wreath of flowers. Hence I call this form of termination the *flower-wreath ending*. The terminal expansions of the axis-cylinders of these nerve-fibres lie on the intrafusal muscle-fibres, but are hardly ever possessed of such long stalks as those of the primary ending.

The Plate Ending.—The size of these endings is extra-

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ordinarily variable. One finds some instances smaller than motor end-plates, some equal in size and some much larger; the last-named are the most usual. There is a difference between these and the ordinary motor end-plates; the terminal expansions of these plate-endings are attached to short and extremely delicate filaments, so that they form, as it were, chaplets, in which rounded axis-cylinders and cross-pieces of the finest delicacy succeed each other in turn. The chaplets which compose a plate-ending anastomose one with another by minute filaments, and at the extreme margin of the plate terminate with obvious enlargements that are rounded off, on this side turned away from the plate. It is usual for a single individual fibre to pass to each plate-ending.

I have not met with each of the above-described endings in every spindle; it is for that reason that it seems to me important to distinguish three forms[1] of "spindles": (1) The spindle with complex nerve-ending; (2) the spindle with simpler nerve-ending; and (3) the spindle with simplest nerve-ending. In spindles of the first class there co-exist a primary form of ending, two secondary forms, and as many as twenty or more of the plate-endings. The primary ending and the two secondary are almost always found very near together; the plate-endings are set at various distances, some in the proximal and some in the distal parts of the spindle.

In spindles of the second class there occur a single primary form of ending, a single secondary form of ending, and a number of plate-endings, set as in the spindles of the first class.

In spindles of the third class there exist a single primary form of ending and a few plate endings, always arranged as in the spindles of the two other classes.

From this it is clear that, firstly, the primary form of ending, *the annulo-spiral ribbon*, is met with constantly in all spindles of the cat; secondly, that the secondary form of ending is that which can vary and even be wanting alto-

[1] Accad. Medico-fisica di Firenze, February, 1896.

gether. Hence the nomenclature I have ventured to propose and adopt.

Of the three classes of spindles examples are not met with equal frequency. In the cat, examples of class 1 are the most numerous; examples of class 2 the least numerous.

To the two older views which considered, on the one hand, that the muscle-spindle was an embryonic centre for the development of new muscle-fibres, or, on the other, a pathological product, we cannot, and ought not, to-day to allow anything more than a historical value. They are opinions which we have had, of necessity, to discard as our knowledge of these special nervous organs has progressed. Sherrington, besides having furnished some valuable additions to our knowledge of the structure of these organs, has succeeded in demonstrating, by means of his experiments, that the myelinate nerve-fibres ending in them suffer no damage after the complete destruction of the anterior (motor) spinal nerve-root, and has proved that the nerve-fibres in question take their origin in the cells of the spinal ganglia. If to this experimental result be added now the new data afforded by my anatomical observations, it will be clearly evident that there is an indisputable basis of truth for the new view that the muscle-spindle is of sensorial nature.

II.—Tendon Organs (Golgi Organs).

In 1892 I called the attention of histologists to two forms of nerve-ending that can be found in the tendon organs. The few observations which I then published I have since largely added to, and have further extended my enquiry to the structure of the tendon of the musculo-tendon organ, with the view of throwing light on certain facts which were laid stress upon by A. Cattaneo and Ciaccio. Together with the nerve-fibre proper to the Golgi tendon organ I find in the cat another much thinner myelinite nerve-fibre accompanying the proper fibre. I call this the *concomitant fibre*. This fibre, arrived in contact with the musculo-tendinous

organ, loses the myelin sheath, and begins to undergo subdivision into a number of secondary fibres, and these latter are so tenous that only a very perfect reduction can reveal them in the preparation. These excessively minute fibres do not anastomose one with another, but they do form a plexiform network, more or less extensive, though never very extensive, because the number of their subdivisions is never very great. Generally they follow a direction from the tendinous end toward the muscular end of the Golgi organ, and terminate close to where the tendon passes into muscle-fibre, or in the muscle-fibre, even at some distance from the tendon of the organ. Their mode of ending consists in the formation of an apical beaded chaplet, the end bead being much larger than the more proximal. This ending sometimes can be seen to be not on the muscle-fibre proper, but on the membrane which Sherrington names the axial sheath, because it lies within his periaxial space. It seems, in fact, to me that these concomitant fibres, with their twisting course outside the Golgi tendon organ, adhere to it rather than strictly speaking penetrate it or belong to it. The existence of the above-described plexiform nerve-ending may be really rare, or its rarity may be only a seeming one, on account of the difficulty of regularly obtaining the exquisite and indispensable delicacy of reduction required to make it visible to the eye of the observer. As to the structure of the tendon of the tendon organ, I have succeeded in demonstrating that the tendon is in reality like an ordinary secondary bundle of tendons. In a transverse section it can be seen that the tendon has two strata, an external and an internal. The former is softer and less compact than this latter. The outer layer is of fibre bundles circularly disposed, and from the inner face of this sheath septa pass inwards, which divides the tendon into two or more compartments. These compartments are occupied by tendinous bundles of longitudinal arrangement. In thus demonstrating the characters of the tendon of the Golgi organ, I find my data easily explain the nature of the *ring constriction* of A. Cattaneo, and of the *fibro-elastic band* of Ciaccio.

III.—Pacinian Corpuscles.

In 1892 I described the existence of Pacinian corpuscles in muscle, and Sherrington has since confirmed my discovery by observations of his own. Some of these Pacinian corpuscles lie near to and bear special relation to the tendon organs of Golgi. Sometimes they lie near the muscular and sometimes near the tendinous end of the organ. They have, for the most part, the same direction as the long axis of the tendon organ. By using serial transverse sections I have been able to get an accurate picture of the relations of these Pacinian corpuscles to the Golgi organs.

Just before or immediately beyond the preterminal constriction the nerve-fibre, which up to that place had been simply lying alongside of the tendon organ, turns and pierces the sheaths of the organ. It however only runs to a short depth into the organ, and then proceeds along it close beneath its surface. It then repierces the sheath partly or wholly, and in an outward direction, and terminates in its own small Pacinian corpuscle. This Pacinian corpuscle lies situate among or on the outermost fasciæ of the Golgi organ. In fact, the small Pacinian corpuscle lies embedded in a hollow scooped out of the surface of the tendon organ. This relationship I have studied especially in the cat. I have not been able to satisfy myself that it exists in the rabbit; in the latter animal, to judge by what I have seen, Pacinian corpuscles lie near to but not integrated in the connective tissue of the tendon organs. Typical Pacinian corpuscles are found in the muscles, but those which lie beside the tendon organs differ from the classical type in volume and in development of lamellated sheath. They are quite small, very elongated, and possess only four to eight capsular lamellæ. The axial portion is always thick, and its nerve-fibre myelinate. There is never more than one nerve-fibre, and that bears a well-developed terminal swelling. In the rabbit these corpuscles are rather longer and more lamellated than in the cat.

Pacinian corpuscles are met with frequently also, not actually inside the muscles but outside, in the perimysium.

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They are here free of all connection with tendon organs, and are no doubt equivalent to the perimysial Pacinian corpuscles well known in the human perimysium since the descriptions of Golgi and Mazzoni.

Finally, I may take this occasion to state the general conclusions that are to be inferred from these results.

There are in the voluntary muscles nerve-fibres of three kinds: motor, sensory, and vaso-motor.

The motor nerve-fibres end, as is well-known, in the *end plates* of Rouget and Kühne.

The sensorial nerve-fibres possess three quite distinct end organs in man and in all the higher vertebrata. These sensorial end organs of muscle are—(1) the muscle-spindles, (2) the tendon organs (or Golgi organs), and (3) Pacinian corpuscles.

The vasomotorial nerve-fibres form reticular plexuses or true terminal plates (Mazzoni), or terminate simply on the capillary walls with a final apical enlargement (Ruffini).

The function of the motor plates and vasomotorial endings has been known for years. Further, physiological experimentation is now wanted to investigate the functional activities of the three sensorial organs—the spindles, the tendon organs, and the Pacinian corpuscles. In my opinion it is upon these three kinds of sense organ that physiology must turn its attention if it will resolve the problem of the muscular sense.

SHORT NOTE ON SENSE ORGANS IN MUSCLE AND ON THE PRESERVATION OF MUSCLE SPINDLES IN CONDITIONS OF EXTREME MUSCULAR ATROPHY, FOLLOWING SECTION OF THE MOTOR NERVE.

BY VICTOR HORSLEY, F.R.S., F.R.C.S.

As a small contribution to the question of existence of sensory nerve endings in muscle and of the preservation of muscle spindles in muscular atrophy I desire to publish the following photographs:—They were obtained from tranverse sections of the gastrocnemii and in some cases the solei of cats and dogs in which the sciatic nerve had been divided, at varying periods before the animal was killed. These periods varied from three days to one year. At the same time I wish to record the existence of Pacinian bodies which I have also found in the same muscles; and to draw attention in passing to the relative degree to which the gastrocnemius in the cat is composed of red and pale fibres mixed.

The Muscle Spindles.

The muscle spindles of the normal gastrocnemius of both the cat and the dog are as a rule so distended in the equatorial region with lymph that the bundle of muscle fibre, nerve fibre, and capillary occupies only about a third to a quarter of the area of the space as seen in tranverse section. This proportionate volume of the components of the spindle is not altered during the first few days of the atrophy, but as a rule by the seventieth day there is apparently a shrinkage of the spindle, such shrinkage, be it remembered, being parallel to the general shrinking which the atrophy of the muscle gradually undergoes as a whole (see photograph,

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section C). At the same time the muscle fibres are apparently wholly unaltered in character. Very exceptionally a muscle spindle may be found in which no collapse or shrinkage of its cavity has occurred, and this even in the most atrophied specimens.

I must now refer to the condition of the muscle in the cases recorded. In the cases I have examined, and of the cat, the muscle fibres lose their diameter, *i.e.*, their volume, by simple atrophic shrinking, the distinction between the red and pale fibres becomes more defined (see photograph, section D), and the striation is preserved almost to the end. In addition to the loss of substance the particular experiments in the course of which these observations were made, seem to show that after a preliminary increase of metabolic activity there is a steady diminution of the same, parallel with the well-known steady decrease in the force and increase in the duration of a contraction. However, even in the most completely atrophied muscle I have been always able to obtain a very slight movement on direct excitation.

Sensory Nerve Endings in Muscle.

In 1883 I showed that in the sheaths of nerve trunks besides end bulbs or tactile corpuscles there were also true Pacinian bodies. I now wish to report that I have found the same within the gastrocnemius muscle in the cat (see photographs, sections A & B). In one case the body (in this instance a double Pacinian body) lay close to one of the inter-muscular septa of the strong aponeurotic fascia of the muscle, but in another case it was buried in the muscle between the bundles of fibres. These Pacinian bodies are ellipsoidal in tranverse section and have diameters of 104 and 144 μ at their thickest part, and present the ordinary well-known structure. The head of the core in one case at least is trifurcated. (See photo.) Whether in the midst of the totally degenerated muscle or in normal muscle these Pacinian bodies show no difference in structure, and do not exhibit any change, which seems to suggest their possessing a high (? central) nutrition efficiency.

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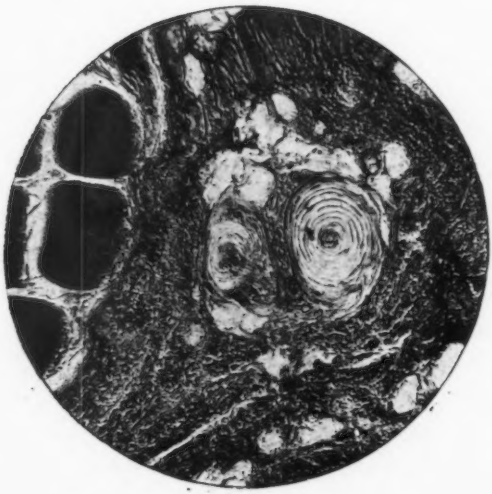
SECTION A.—Tactile body in normal muscle.



SECTION B.—Tactile corpuscle (double).

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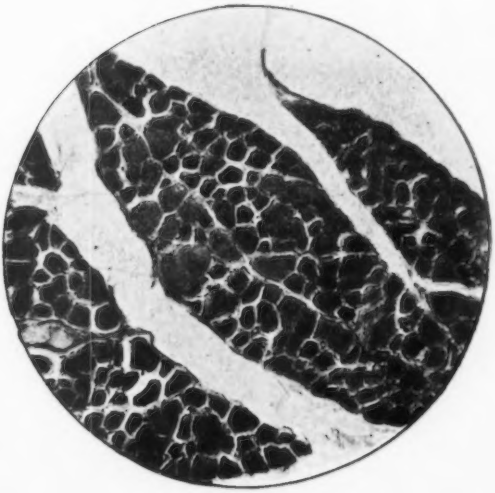
SECTION C.—Shrinkage of muscle spindles.



SECTION D.—Pale and dark muscle fibres.

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A CASE OF ACUTE GRAVES' DISEASE, WITH A DESCRIPTION OF ITS MORBID ANATOMY, AND OF A SERIES OF MICROSCOPICAL SECTIONS.[1]

BY ARTHUR FOXWELL, M.A., M.D., F.R.C.P.

Physician to the Queen's Hospital, Birmingham, and Examiner in Medicine at Cambridge University.

ANYTHING which tends to elucidate the obscure ætiology of Graves' disease is worthy of note. The following case presents a pathological state of the base of the brain not usually described in this condition. This is my excuse for venturing to place this imperfect report before the Section.

Rachel W., 41, was admitted into the Queen's Hospital, Birmingham, on June 21, 1893, complaining of weakness, anorexia and rapid wasting. Her parents were alive and healthy and possessed a comfortable home; four sisters and one brother were also of good constitution; one sister died in childbirth, and two brothers in infancy. There was no history of neurotic tendency in any near relative. Her previous health had also been quite good, except that, ten years ago, she strained her left side whilst rowing and had suffered occasional pain there ever since. There was no suspicion of syphilis or alcoholism. Her present illness commenced at Christmas, 1892, six months prior to her death. Her first symptoms were weakness and a stiffness of the joints on rising in the morning. Except for the slow increase of the weakness and a gradual onset of dyspnœa on exertion, she remained fairly well till March, when she began to suffer from morning nausea without vomiting. In April she first noticed an enlargement of the thyroid, the increase was at first rapid, but it has since varied much in size, sometimes the fulness of her neck

[1] A paper read in the Section of Pathology at the Annual Meeting of the British Medical Association, in August, 1895.

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almost vanishing for a week at a time so that she could easily fasten her dress. At this time, too, Dr. Quirke, of Handsworth, first noted undue prominence of the eyeballs, this she had never observed herself though her friends had done so.

For a month previous to admission she had suffered from vomiting and for a fortnight she had been unable to take solid food, though she could not say she had any difficulty in swallowing. For a fortnight she had been too weak to walk; and had perspired freely whenever she fell asleep.

On admission she was found to be a spare but well-formed woman of average size, nervous temperament and dark complexion. She appeared greatly emaciated. Her mentality was quite clear, though there seemed to be much anxiety and mental tension. There was extreme restlessness of one or other portion of the body, though as a whole the body remained still, maintaining the dorsal decubitus. Her cheek bones were prominent, she had a hectic flush, and the temperature was 99·4°. The pulse was 120, small, regular and of low tension; the radial felt healthy. The respirations were 32 to the minute and rather panting in character. She was without appetite, an unpleasant taste being constantly present. The tongue was very raw and vividly red; it was deeply fissured and had scattered patches of dirty fur. The eyes were prominent, but not strikingly so, the sclerotic not being visible above the cornea. The left pupil was larger than the right.

The Thyroid had, just to the right of the mid line, a rounded, hard, walnut-sized nodule; beyond this was a moderately enlarged right lobe over which a systolic murmur was plainly heard. The left lobe was also slightly enlarged. There were no pressure symptoms. The cardiac impulse was best felt in the fourth space within the nipple line, but it was largely diffused from without the nipple line in the fifth space up to the second space close to the sternum. There were pulmonary, tricuspid and mitral systolic murmurs; and, in the veins of the neck, a loud *bruit du diable* with marked systolic pulsation, especially during inspiration. Friction sounds, cardiac in rhythm, were heard over the sternum between the third and fourth cartilages. In the first left space and, to a less extent, in the second space was a curiously circumscribed and definite area of deficient resonance; so well marked was it that my able house physician, Mr. J. A. H. White, and myself, decided that it could only be due to an enlarged thymus gland. At the apex of the right lung the percussion note was slightly deficient and the

expiration lengthened; whilst in the right interscapular region the breath sounds were feeble. Otherwise the lungs appeared healthy. The abdomen was convave and rather hard, the iliac crests being very prominent. The hepatic and splenic dulnesses were normal and neither organ was felt. The urine was fairly normal in quality and quantity.

Treatment and Course of Case in Hospital.—It being very difficult to administer food by the mouth, the patient was fed by nutrient enemata to which was added three times daily a ♏ 40 dose of bromidia, and she was given in addition one teaspoonful of milk every half-hour. On the 24th, as the rectum was exceedingly irritable, two enemata only were given daily, by the long tube, to each of which was added ♏ 20 of laudanum instead of the bromidia. The nausea was now excessive, and a vomit of clear, greenish, strongly alkaline fluid became frequent. On the 25th the patient stated that she had no nausea, but could not avoid almost constantly regurgitating her food. On the 26th she was given ♏ j. of ipecacuanha wine every hour and had no more regurgitation till shortly before death.

On the 27th the pulse rate rose to 168. The restlessness increased. There was a tendency to bed-sores, and "on the hands and feet was slight desquamation." The temperature which had varied from 98·5° to 99·5° now took the higher level of 100° to 101° and so remained till death. On the 28th, 35 ounces of saline fluid were injected into the subcutaneous tissue of the axilla. The pulse, which had been quite uncountable, improved and the restlessness became somewhat less. The next day the pulse and restlessness again grew worse; the emaciation was strikingly rapid; her thirst painful to witness; nothing of any kind could be retained when given by the mouth; and she died exhausted, early on the morning of the 30th.

Briefly, then, the salient features of the case were these:

(1) The three cardinal symptoms of Graves' disease.

(2) Enlargement of the thymus.

(3) Dilatation of the left pupil.

(4) Moderate pyrexia, apparently due to the lung.

(5) Extreme restlessness, rapid wasting, uncontrollable vomiting which was sometimes without nausea or retching. Clinically, it was these last which occupied our attention and made us think there must be some cerebral lesion, the gastric irritability being due to involvement of the vagus.

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(6) The rapidly fatal issue, only two months after the enlargement of the thyroid was first noticed.

The post-mortem examination was made thirty hours after death by our pathologist, Dr. Kauffmann, to whom I am indebted for the following excellent report.

The body was rather emaciated with much wasting of the subcutaneous fat.

The thymus extended from the lower border of the thyroid isthmus to the second intercostal space, overlapping the upper part of the pericardium; it was one and a half inches wide, but so thin that it did not weigh half an ounce.

The central portion of the upper lobe of the right lung was of a dirty red colour and contained distinctly less air than the rest. The front border of the right lung was also large and emphysematous. Otherwise the lungs were normal.

The heart was not enlarged, nor was there pericarditis. The aorta was full and distinctly pushed the pulmonary artery to the left. The apex lay beneath the fifth rib, one inch without the costo-chondral joint. The right auricular appendage reached up to the lower border of the second left cartilage. The valves and muscle were healthy.

The kidneys were healthy. Of the liver there is no note, but our memory is that it was healthy. The spleen was small, hard and granular.

The uterus was retroflexed; at its hinder part was a fibroid tumour as large as the body of the womb.

Of the thyroid, unfortunately, there is also no note, but we believe it was in the condition usually found in Graves' disease.

The ganglia of the cervical sympathetic were rather small and wasted throughout, but not harder than normal.

Only the upper portion of the cervical region of the spinal cord was examined; this appeared healthy.

The brain was slightly more adherent to the skull-cap than normal; its surface veins were full, both in front and behind; the arteries at the base were also tolerably full.

There was a patch of yellowish fibrous thickening over

the foremost part of the vermiform process of the cerebellum; this was small in extent but was surrounded by a distinctly thickened, and slightly milky patch of pia of much greater extent. The arteries and veins, chiefly on the upper surface, were markedly full and rather more tortuous than usual.

The third ventricle. While the front commissure appeared fairly normal, the middle and hind ones were much altered. The middle commissure was represented anteriorly by a very thin, grey, gelatinous mass of nerve tissue, no thicker than an ordinary sewing thread; whilst behind this, replacing what should have been the bulk of this commissure, were several delicate, but firm and strong bands of fibrous tissue connecting the adjacent surfaces of the optic thalami, which at these points were thickened, puckered and irregular. The hind commissure appeared thin and supported a dark grey pineal gland which was very friable (owing perhaps to *post-mortem* change).

On the upper part of the opposed surfaces of the optic thalami were two patches of grey translucent softening; these did not extend deeply into the substance of the thalami. The choroid plexus was not congested.

The internal capsule and its surrounding parts, except for an undue number of puncta cruenta, as well as the iter and corpora quadrigemina, appeared healthy. So also did the pons and cerebral hemispheres.

The fourth ventricle presented a remarkable appearance. The whole floor looked thickened and the finer shadings were lost, but the natural hollows were more marked than normal:—*e.g.*, the two depressions on either side of the middle line in front of the auditory striæ which striæ were quite absent. The calamus scriptorius itself was too deep and too sharply marked. The whole looked as if the more delicate distinctions had been swallowed up in an exaggeration of the main lines and a thickening generally of the tissue.

For the microscopic sections of the medulla and pons which I am enabled to show you, I am indebted to the kind skill of Dr. Douglas Stanley, Pathologist to the General

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Hospital,[1] and it is with his aid that I have written the following brief account of the appearances noted in them:

(They were all prepared according to the Weigert-Pal method—that is, the material was hardened in Müller's fluid. The sections were stained in hæmatoxylon and then bleached by being placed in a $\frac{1}{4}$ per cent. solution of permanganate of potassium, and afterwards in a solution of $\frac{1}{2}$ per cent. pure oxalic acid and $\frac{1}{2}$ per cent. of sulphite of potash. They were then washed in distilled water, and afterwards in tap water.

The result is to stain the medullated fibres blue, the nerve nuclei a brownish yellow, whilst the neuroglia and other fibrous tissue is almost white.)

The lowest section (*Right hand figure of No.* 6 *slide*) is through the lower end of the olivary body ($\frac{1}{12}$ in. above the horizontal line *d* in Obersteiner's figures). The olivaries themselves were healthy. The vessels are dilated and engorged throughout but especially on the left half. The tissue around the central canal takes the stain badly and has an indistinct, ground-glass appearance; in it are several "amyloid" bodies.

The next section (*Middle figure*) at the level of the Calamus is similarly gorged. The pia is much thickened, and on each side of the central cleft of the floor of the fourth ventricle, it is indistinguishably mixed with the gracilis column; the differentiation of nerve elements being almost effaced, whilst the production of new blood vessels is great.

The third section has still marked congestion though slightly less than the preceding. There is the same thickening of the pia on the floor of the ventricle, and here it passes into and involves the nucleus of the gracilis. The pia and the underlying tissue together form a sclerosed layer about six times the thickness of the normal membrane. The nucleus of the twelfth nerve as here seen is normal.

The fourth section (*Slide* 5, *right hand figure*) at the level of Obersteiner's line *e*, shows the nucleus of the tenth nerve involved in the sclerosis, though to a less extent than

[1] Now physician to out-patients at the Queen's Hospital.

was that of the gracilis, for its nerve cells appear to be but little changed. The ascending root and nucleus of the eighth and the nucleus of the ninth show no morbid change.

Number 4 *slide* (sections 6 and 7, at level of Obersteiner's lines *g* and *h* respectively). The vessels of the pia are much distended and there are one or two spots of hæmorrhage. The pia is rather less thickened, but its thickening extends over a wide area.

Slide No. 3 (with sections 8 and 9 respectively, at levels between Obersteiner's lines *h* and *i*, and *i* and *k*), shows a large excess of vascularity in the nuclei of the eighth and seventh nerves, especially on the left side; but there are no definite hæmorrhages. All the central region of the cord in these two sections has also an excessive vascularity. The pia is still thickened, but now the thickening is less on the median raphe of the floor than at either side.

Number 2 *Slide* has sections 10 and 11 at levels a shade higher than Obersteiner's *m* and midway between his *m* and *n* respectively. The lower section shows several well-marked capillary hæmorrhages beneath the floor of the fourth ventricle on each side of, and in, the median line. Corpora amylacea exist over a wide area, occupying the whole of the tegmen and also extending into the pyramids.

The higher section shows nothing remarkable except excessive vascularity.

Slide No. 1, Section 12, just below the corpora quadrigemina, at Obersteiner's line *o*, shows less vascularity. There is an occasional hæmorrhage but otherwise nothing of note.

In brief, then, the changes found were:

(1) Chronic inflammation-sclerosis of the pia covering part of the vermiform process and of that covering the floor of the fourth ventricle. Also of the middle commissure of the third ventricle.

(2) Acute softening of the surface of both thalami.

(3) Excessive vascularity of the surface of the brain, of the internal capsule, of the cerebellum and of the medulla—in the medulla, at any rate, leading to occasional hæmorrhages.

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(4) Sclerotic changes involving portions of the tegmen, the pyramids, the gracilis and their nuclei; and the nuclei of the tenth nerves.

It is difficult to definitely connect any of this morbid anatomy with the clinical appearances manifested by the patient. Bias, no doubt, would tend to make my own imagination run away with me in seeking for cause and effect. Still, the changes described are organic and decided and of sufficient importance, I think, to warrant my bringing the case before this Section.

TRAUMATIC NEURASTHENIA AND HYSTERIA.[1]

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WHEN your Secretary asked me to read at this meeting a paper upon the functional disturbances of the nervous system following accidents, I promptly asked the privilege of changing the title to something more definite. Thanks to the new methods of research the old distinction between functional and organic diseases is fast disappearing, but, were it still maintained in its old-time definiteness, it is a question as to what or how many of the more obscure traumatic nervous affections should be classed under either heading. It was once thought that most of them were due to more or less pronounced changes in the spinal cord, such as we used to call organic, and later it was held by many that they were all of a functional character, but now the current of opinion (1) seems to tend to the belief that a part of them are due to organic changes, although the greater part are probably dependent upon those slighter changes which we call functional, and which can be detected, if at all, only by the recently discovered methods of staining.

Next to the old term of "spinal concussion," no name seems better calculated to add obscurity to the obscure than the new term of "traumatic neurosis." The word neurosis, of itself, is so often understood to mean functional nervous disease that it has a misleading significance. Traumatic neurosis, too, may designate certain cases of chorea, epilepsy, cephalalgia, or paralysis agitans, as well as hysteria and

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other obscure affections. It has added much to our ignorance of mental diseases, for example, to find that ætiology, symptoms, pathology, course, diagnosis, and prognosis have been studied from statistics of cases of amentia, general paralysis, mania, melancholia and paranoia all classed together and regarded almost as a single morbid entity, "insanity." The employment of the term "traumatic neurosis" is almost as confusing. It is only by a careful differentiation of the various affections, and a study of such disorder separately, that we can attain definite knowledge. It may still be impossible to draw a sharp boundary between hysteria and neurasthenia, as it is to draw one between tabes and paretic dementia; it is possible that in the future we may find that there are several distinct affections, all of which we now term hysteria; but, in spite of errors and ignorance, it is better to classify so far as we are able rather than to accept the vague and general term of traumatic neurosis.

Among the commoner traumatic affections of the nervous system, especially in cases where litigation is involved on account of claims for damages, are hysteria and neurasthenia. To determine this point I have classified two hundred cases of the more obscure traumatic nervous diseases seen in hospital and private practice. I have included all the litigation cases of traumatic nervous disease of whatever sort seen by me during that period, but I have not taken into account, among the non-litigation cases, those cases of injury to the peripheral nerves, meningeal hæmorrhage, fracture of the skull or spine, epilepsy, &c., where there was no doubt as to the nature of the trouble. As my object was primarily the study of the obscurer forms of nervous disease such omissions were justifiable, but the percentages here given are too great if we include all cases due to trauma.

Of these two hundred cases one hundred and three were not, so far as I know, the subjects of litigation, and a few of the ninety-seven litigation patients consulted me after all litigation was over simply for advice and treatment, and not to obtain my opinion for use in the settlement of their claims. Seventy cases were classed as hysteria and fifty as

neurasthenia, sixty per cent. of the whole. Of the litigation cases fifty were hysteric and twenty-one neurasthenic, eighty-one per cent. of the whole. Of the non-litigation cases twenty were hysteric, and twenty-nine neurasthenic, thirty-nine per cent. of the whole. The importance of these two affections is therefore evident.

Although injuries of the peripheral nerves and the spine or spinal cord are not uncommon in hospital practice, I have very rarely seen them—and my experience is shared by my colleagues in Boston—among the litigation cases. The only explanation that I can find of this is that, as the diagnosis and prognosis in such cases are usually plain, the opinion of a specialist in nervous diseases is seldom required, and the case, if the liability be admitted, is settled out of court.

The preponderance of neurasthenia in the litigation cases is not very great, and it may be due simply to chance, but the greater percentage of hysteria is another matter, and is worthy of consideration. It at once suggests and seems to strengthen the familiar plea that litigation and the desire for gain are important factors in the genesis of these so-called traumatic affections, producing, under the influence of suggestion, a set of symptoms, real perhaps in themselves, but not the result of the physical injury. Strümpell (2) has lately reiterated this plea with much emphasis. Having for some time urged that the chief factor in the production of "traumatic" or "accident neurosis" was psychical rather than physical, he now goes a step further and considers the chief psychical factor to be the ideas of personal gain which arise as a consequence of the accident. "Shall I obtain the accident pension? How much shall I get? How much am I injured in my working capacity?" With but a slight change in phraseology to suit the differences in our laws, these questions may be asked here. "Shall I get damages? How much shall I get? Shall I be able to earn anything afterwards?" We know well all the maleficent psychical influences which may attend these cases. The desire for gain, the necessity of putting the worst foot foremost, the depressing suggestions, the educating examina-

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tions, the worry of litigation, the gloomy prognostications, the law's delay, and, sometimes, the total lack of proper medical treatment during the whole period of litigation put the patient into the worst possible condition and aggravate every symptom. It is no wonder then, that we ask if the physical injury has had anything to do with the subsequent trouble.

Although the whole subject of the so-called "traumatic neuroses" has been much discussed in England, France and Germany, it has awakened very little interest, apparently, south of the Alps (3). If, then, there are no laws in Italy whereby an injured person may recover damages, it may be, granting the truth of the hypothesis, that litigation is the important factor, that the cases are rare in that country, and that, therefore, little attention has been paid to the subject. In answer to my inquiry I have learned from Dr. Seppilli that there are no special laws in Italy relating to railway accidents. When accidents do occur (which is only very seldom the case) the railway companies indemnify, according to the cases, the persons or families for the damages received. The physicians of the company, however, must give their judgment upon the case.

In spite of all this I am disposed to doubt Strümpell's dictum, and to lay much less stress upon these ideas of personal gain and the maleficent psychical influences above referred to in the genesis of the hysterical or neurasthenic symptoms. In the first place, precisely similar conditions have developed in the non-litigation cases, where all these factors are absent; and, in the second place, the symptoms have developed almost immediately after the accident in many of the litigation cases, before these factors could be brought into account. When the symptoms follow immediately upon a period of unconsciousness, profound surgical shock, or marked mental confusion, it is hardly credible to suppose that the first idea to come with the regaining of consciousness is "Shall I get damages?" even though the lawyer's "runner" has reached the scene of the accident before the ambulance. That these factors may have a very bad influence in aggravating or prolonging the disease is

readily conceivable and most probable, but they certainly do not always create it.

I have just referred to the similarity between the litigation and the non-litigation cases. To determine this more accurately I have carefully tabulated the symptoms presented by these two classes of cases, both of hysteria and of neurasthenia. The relative frequency of the symptoms in the two classes of cases was about the same, and the rarer symptoms, observed perhaps in a single case, occurred now in one class and now in the other, as chance ordained. A comparison of individual cases in the two classes showed no striking differences. In other words, from the evidence presented by these hundred and twenty cases of hysteria and neurasthenia, seventy-nine of which were litigation cases and forty-one of which were not, I can find no symptoms which would warrant a diagnosis of "litigation hysteria" or "litigation neurasthenia," as distinct from "traumatic" hysteria or neurasthenia, and no essential difference between the two classes of cases, with one exception. In many, but not in all, litigation cases the patients have dwelt upon and have worried over the prospective law-suit. Worry and the dwelling upon certain ideas are, however, common in neurasthenia and hysteria, no matter what the cause, and in these cases the law-suit has merely furnished the special subject for worry.

It is a fact, however, that these non-litigation cases of hysteria, but not of neurasthenia, have been, as a rule, of a less severe type and have progressed more favourably than the litigation cases. This may be due to the absence of those injurious psychical influences attending litigation, to which I have already referred, and the more prompt inauguration of systematic treatment, but there are other factors which I believe have exercised a marked influence upon the severity of the manifestations.

That hysteria and neurasthenia may be due solely to psychical causes is a well-accepted fact. Even in traumatic cases the physical injury may be insignificant and the psychical shock the only factor. I have seen, for example, a marked somnambulic state develop from spraying the throat.

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In emphasising the psychical factor, however, I believe that Strümpell and others have gone to an extreme, and have not given due weight to the physical injury. In order to estimate the importance of such physical injuries, and to find some reason why the percentage of hysterical cases should be so much greater in the litigation than in the non-litigation cases, I have studied the nature of the accident in these hundred and twenty cases.

Of the fifty litigation cases of hysteria here classified, seven were due to collisions of street-cars, seven to being thrown from a carriage, seven to falls on getting on or off the cars, six to railway accidents (in four cases of a grave character), six to electrical shocks, five to elevator accidents, three to falls, and two to blows on the head. Of the twenty non-litigation cases, six were due to falls, four to being thrown from a carriage, two to blows on the head, and two to blows on the arms. Of the fifty litigation cases, eighteen received severe physical injuries and two severe electrical shocks, while sixteen received moderate physical injuries and two moderate electrical shocks; in five cases there was a fracture of some bone. Of the twenty non-litigation cases two received severe, and ten moderate, physical injuries; in no case were any bones broken. From the nature of these accidents, as thus hastily outlined, it may be inferred, and the inference will be confirmed by detailed study, that the psychical factors of fear, horror, excitement and the like, were much greater in the accidents of the first class than in those of the second. In the majority of the non-litigation cases the accidents were of a simple character, unattended with any special terror or scenes of distress; in the litigation cases alarming features were not uncommon.

Turning now to the cases of neurasthenia, I have found that the severity of the symptoms has been, on the average, about the same in the two classes of cases. Of the twenty-nine litigation cases, six were due to railway accidents (in five cases of a grave character), five to falls on getting on or off cars, five to falls, four to blows on the head, and three to being thrown from a carriage. Of the twenty-one non-litigation cases twelve were due to falls, two to being thrown from a carriage, two to blows on the head, and two to strain.

Of the twenty-nine litigation cases eight received severe and nine moderate physical injuries, and one a moderate electrical shock; in three cases bones were broken. Of the twenty-one non-litigation cases four received severe physical injuries, one a severe electrical shock, and eleven moderate physical injuries; in three cases bones were broken.

From these facts it seems possible that, apart from any questions of litigation or suggestions arising from the desire of gain, there were reasons why there should be a larger number of cases of hysteria in the litigation series, and why they should be of greater severity. Litigation and the desire for gain are also present in the neurasthenic cases, but the severity of the accidents was about equal in the two classes, and we find a corresponding equality in the number and severity of the cases. The only conclusion, therefore, to which we can come is that litigation and the desire for gain play only a subordinate part in the genesis of these morbid conditions, and that the physical as well as the psychical shock of the accident is a very important factor.

I have already said that psychical shock alone may produce hysteria or neurasthenia—a fact repeatedly demonstrated by the researches of Guinon (4), Janet (5), and Breuer and Freud (6). The question naturally arises, can physical shock alone act as a cause? A satisfactory answer cannot be given. Even in the simplest accident, a fall on the ice, a strain in lifting, or a sharp blow on the arm against a piece of furniture, there may be some element of fear or apprehension to arouse a strain of morbid mental conditions. When a man is rendered unconscious by a sudden blow on the head, and later has no memory of the accident, it may be that, in the interval "between the saddle and the ground" on which the theologians once laid stress, there was some suggestion of future evil which hypnotism might reveal. One of my colleagues, a young physician, who in his hospital life had become somewhat familiar with accidents and scenes of distress, has suffered for a year and a half from neurasthenia, the result of a serious railway accident in which several persons were injured. He felt a jerk, lost consciousness for a second, and recovered his senses

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to find himself standing in the aisle of the car facing to the rear, while the car next behind was crashing into the one he was in. He was uninjured and spent some time at the scene of the accident caring for the wounded. He stated that he was not frightened at all, and doubted whether there was any period of momentary fright of which he was not conscious. The sensation was exactly as if he had been struck a very heavy blow in the back of the neck with a sledge hammer. Such a statement from a trained observer I believe to be of some importance in deciding the question, and my own belief is that in a part of the cases the psychical shock, although it can not be wholly excluded, is distinctly a subordinate factor in the genesis of the trouble.

Of the other factors in the ætiology it is hardly necessary to say very much. With regard to age four of the cases of hysteria were under twenty, one fourteen, two sixteen, and one (the only litigation case) eighteen. Three neurasthenics, all of them litigation cases, were under twenty, one seven, one fourteen, and one seventeen. Women are much more subject to these affections than men. Thirty-eight men out of one hundred and thirty-seven (twenty-eight per cent.) had hysteria, and twenty-seven (nineteen per cent.) had neurasthenia. Thirty-two women out of sixty-three (fifty-one per cent.) had hysteria, and twenty-three (thirty-six per cent.) had neurasthenia. Thirty-seven out of forty litigation cases and eighteen out of twenty-three non-litigation cases among women had one or the other of these affections. A neuropathic heredity, upon which the French especially have laid much stress, I have found to be of minor importance. I have obtained a history of a questionable heredity (nervous disease, tuberculosis, &c.) in eight cases of hysteria and seven of neurasthenia. A previous history of poor health was elicited in twelve cases of hysteria and ten of neurasthenia. A history of previous syphilis or of abuse of alcohol was only rarely obtained. Many of the litigation patients, however, might be disposed to conceal any hereditary or pre-existing weaknesses.

Traumatic hysteria and traumatic neurasthenia have many symptoms in common, so that the designation of the

former affection as hystero-neurasthenia is not unwarranted, and the boundary between the two affections is not very distinct. Headache, nervousness and irritability, depression, insomnia, vertigo, failure of memory and mental power, pain in the back and limbs, paræsthesia, palpitation, dyspnœa, a rapid pulse, loss of appetite, dyspepsia, constipation, diminished endurance, inability for physical or mental application, general muscular weakness, and exaggerated knee-jerks, are common to both.

Of the symptoms common to the two affections a few are worthy of note on account of their objective value. In over one-half of the hysteric cases, and in over one-third of the neurasthenic cases the knee-jerk was distinctly exaggerated, being often attended with the patellar twitch and front-tap contraction, and very rarely with a spurious ankle clonus.

In a large majority of the cases, both of hysteria and neurasthenia, there was a decided increase in the rapidity of the pulse, it being over eighty and frequently weak, irregular or intermittent. Congestion, coldness of the extremities, flushing, excessive or localized sweating, and other vaso-motor disorders were not uncommon, but œdema was rare and cardiac murmurs and enlargement of the heart were exceptional. In a very few cases there was an abnormally slow pulse.

Mannkopf's test, the increase in the rapidity of pulse on pressure upon an alleged tender region, I have found of distinct value. Instead of an increased rapidity the pulse may become weaker or irregular under such conditions. In forty-five out of forty-nine cases the test has given definite information, but when there was other conclusive evidence of the disease this test, on account of its painful character, was not employed. Not only is it of value as a test of tenderness, but it is also a test of muscular effort. When a patient executes a slight movement, lifting the leg, closing the hand, stooping forwards, only by an apparently great effort, if we find that the pulse is materially quickened we have proof that the effort really was great. Flushing of the face and an outbreak of sweat may also be noted under such conditions. The test may also be used for analgesia. If the

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pulse rises in consequence of a painful stimulus in a healthy area and does not rise when the same stimulus is applied to the alleged analgesic area, we have proof of the genuineness of the analgesia.

The chief distinction, so far as our present knowledge goes, between neurasthenia and hysteria is to be made by the detection of the various hysterical stigmata.

Of the various hysterical stigmata pronounced amnesia, after the initial period of amnesia due to shock, or pronounced aboulia was distinctly rare. An enfeeblement of the will power and a failure of memory corresponding to the general asthenic condition can hardly be classed as distinctly hysterical. Hallucinations of sight and hearing, temporary delirium, and temporary confusion were occasionally met with both in hysterical and neurasthenic cases, usually in the period immediately following the accident. Attacks of one sort or another were noted in sixteen cases, twelve of the litigation cases and four non-litigation cases. In none of these cases did these attacks manifest the typical features of the "grand attack" in its four stages, but occasionally they were severe enough to show the *arc de cercle* or other striking phenomena. In no case could I detect any hysterogenous zones. Hemiplegia was noted in twenty-five cases, menoplegia in ten, and paraplegia in one, contractures in five, tremor in twenty-four, ataxia in eight, mutism in three, and somnambulism in one.

The most frequent stigmata were those in the sensory domain. Hypoæsthenia was noted in sixty-four per cent. of the cases, analgesia in forty-nine per cent., and distinct anæsthesia in twenty per cent. Thermoanæsthesia was less often sought for, but was noted in seven out of thirteen cases. Many patients, fifty-seven, complained of numbness or other abnormal sensations, chiefly in the affected area (7).

In a few of the cases presenting other hysterical stigmata no disturbances of the cutaneous sensibility were manifest, but in most of these cases the examination was hurried and the opportunity for prolonged observation was lacking. Of course, in rare cases hysteria may occur without any disturbances of cutaneous sensibility or any contracture of the

visual field (8). In a considerable number of cases the loss of tactile sensibility was very slight. The patient could feel the slightest touch upon the affected area of the skin, but it was not felt quite so distinctly as on the corresponding healthy area. It required careful examination to detect the difference, so that in many cases it might be readily overlooked. In some very severe cases there was only this slight difference. In some cases the affected side is distinctly colder to the touch, especially in the hand or foot, and the patients complain of an increased sensitiveness to cold. Further valuable information, as confirming the genuineness of the sensory disturbance, is afforded by the condition of the cutaneous reflexes. The plantar reflex is frequently diminished or lost on the anæsthetic side, and the other reflexes sometimes show a similar change. Rosenbach long ago held that the abdominal reflex was not affected in cutaneous anæsthesia, unlike the other skin reflexes, but this is incorrect, as in no less than four cases it was diminished or lost on the affected side, and in one case it was diminished on one side when the plantar reflexes remained equal. The knee-jerks, too, often show an inequality, but this seems to have less relation to the side affected. It is rather more apt to be exaggerated on the affected side, as seen in eight cases, but in five cases it was exaggerated on the sound side.

As is well known analgesia may be present when the cutaneous sensibility is normal, so that many cases may be stated to show normal sensibility merely from lack of thorough examination. I have found the best test for sensibility to pain to be by means of the faradic current, applying Erb's cutaneous electrode to different regions of the skin, and measuring the strength of the current required to produce pain by Edelmann's faradmeter. A current of fifteen to twenty volts will usually cause pain in normal areas, while pain will be felt in analgesic areas only with a current of from thirty-five to eighty volts.

With regard to the distribution of these disturbances of cutaneous sensibility, in forty-two cases one side of the body was affected, but in many of these cases the area of anæs-

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thesia was not sharply defined nor did it affect the complete half of the body; the face was often exempt, the anæsthesia did not reach fully to the median line, and there were some areas on the affected side where sensation was as good as on the healthy side. In eight cases the anæsthesia was confined to a single limb. In two cases the legs alone were affected. In six cases both sides of the body were affected. In one case the anæsthesia was very variable, now occurring in irregularly distributed areas, now affecting nearly one-half of the body, and, a day or two later, shifting to the other side. In a part of the cases the special senses were affected on the anæsthetic side, in some cases only one or two of the senses, and in others they were unaffected. A diminution in the sense of smell on one side was noted more frequently than of the other special senses. Achromatopsia or dyschromatopsia was noted in a few cases, but was not observed in others.

Contraction of the visual field was noted in forty-four cases, but in no case of examination with the perimeter was a normal field found, and in several cases examined in other ways, where the field was apparently normal, the perimeter subsequently showed some contraction, so that it is fair to suppose that contraction of the field is one of the most common sensory stigmata. In only a few cases, however, was the contraction marked, usually the field extending to about forty degrees on the nasal side and seventy on the temporal. The "shifting type" of field, although often sought for, was only rarely found. Reversal of the colour fields, also, was only rarely noted.

In accepting these stigmata as the distinctive symptoms of hysteria as opposed to neurasthenia, we are not free from the possibility of error. In the first place the stigmata may not appear until some time after the injury. In a case at present under observation the visual fields were normal, the cutaneous sensibility unimpaired and there were no motor symptoms five months after the injury; the only possible stigma was a loss of smell on one side. Two months later, however, contraction of the visual fields with peripheral exhaustion ("shifting type") had developed, with right

hypoæsthesia and analgesia, and these symptoms have persisted, with others, for nearly a year and a half. Furthermore, with the progress of hysteria, an existing anæsthesia or hypoæsthesia may wholly disappear, leaving behind only a slight analgesia which may readily escape notice unless careful electrical measurements be made; and finally, all disturbances of cutaneous sensibility may be wholly wanting. In some of these cases the visual field may remain contracted, but in others it may enlarge to normal. It may thus happen that, in ignorance of past conditions or of the future development of the case, we may regard a case as simply neurasthenic that in the past or in the future had shown or will show clear evidences of hysteria. Whether a contraction of the visual field, without any other stigmata, is sufficient to warrant a diagnosis of hysteria, is still open to question. Charcot (9), on the one hand, holds that it does not occur in uncomplicated neurasthenia, but Wilbrand and Sänger (10), on the other hand, whose work on the visual field is of great value, and also Löwenfeld (11), claim that it may occur in such cases. In four cases which I have classed as neurasthenic, there was some contraction of the field without other stigmata of hysteria, and in the absence of other stigmata I have not ventured to class the cases as hysterical. It is true, however, that in non-traumatic neurasthenia I have not yet found contraction of the visual fields, but a careful perimetic examination was only rarely made.

Contracture of the visual field, by itself, may undoubtedly occur in various affections. I have noted it in cases of brain tumours without as well as with optic neuritis, in the early stages of optic atrophy with as regular an outline as in hysteria (contrary to Charcot's statement (12) as to the fields in tabes), in spinal sclerosis, in tobacco amblyopia, in oculomotor paralysis, in melancholia, and rarely in epilepsy, not as a transitory phenomenon following the attack (13), and in other cases where the idea of hysteria was not entertained. This fact, however, is well recognized, and it has been fully demonstrated by the researches of Wilbrand and Sänger.

When, in a given case, we find disturbances of the cu-

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taneous sensibility, not following the distribution of the peripheral sensory nerve areas or the spinal root areas, associated with contracture of the visual field, are we justified in assuming that the case is one of hysteria? If the sensory disturbance affect one half of the body the probability of hysteria seems greatest. Patrick (14) has laid considerable stress upon a sharply defined boundary of the anæsthetic area as characteristic of hysteria. This may be the case, and in a very few cases it was noted, but in the great majority of cases, including many that were undoubtedly cases of hysteria, this sharp outline did not exist. Charcot (15) has claimed distinctly that in hysteria alone do we find profound hemianæsthesia involving the special senses, and Gilles de la Tourette (16), following Charcot's teachings, holds that hemianæsthesia is due either to a capsular lesion or to hysteria. Of course, instead of a capsular lesion there may be a focal lesion elsewhere in the sensory tract. The wider knowledge of hysteria, due to the investigations of the French neurologists, have rendered the conclusions of Thomsen and Oppenheim (17) somewhat unsatisfactory, and further investigations are desirable. If in alcoholism, lead poisoning, multiple sclerosis or epilepsy, we find hemianæsthesia with contracted fields it may often be true that there is a coexisting hysteria, but it is scarcely logical to claim that all such cases are probably hysterical (18), or boldly to assert with Souques (19) that because the patient presents these stigmata he is therefore hysterical. It is not long since it was held that hysteria and syringomyelia were very often associated, because the sensory disturbances in syringomyelia did not follow the regular spinal distribution, but later inquiry has shown that the distribution of anæsthesia in lesions of the cord itself is very different from that in lesions of the spinal roots. It is not at all improbable that careful research will prove that hemianæsthesia is not pathognomonic of hysteria. In migraine I have occasionally heard patients complain of a subjective feeling of numbness over one half of the body during the attack, and Dr. J. J. Putnam tells us that he has noted an actual diminution of sensibility on one side during an attack of supraorbital

neuralgia. I have also seen hypoæsthesia over one half of the back in a case of mitral disease and phthisis. Hemianæsthesia in focal lesions of the brain is, of course, not uncommon. Not long ago I saw for a few moments only a man who had had epileptic seizures in childhood. Three years before he was thrown from a wagon in a collision with a railway train, and after that he was no longer able to do hard work. Two years later the fits returned. When I saw him he had atrophic paralysis of the right shoulder and upper arm, tremor of the hands, exaggerated knee-jerks, and right hemianæsthesia. Not long after he died of pneumonia. The autopsy showed a very peculiar and extreme atrophy of the brain cortex, of unknown origin, involving the whole left ascending parietal convolution and the upper part of the right ascending parietal, but not extending beyond those convolutions.

It becomes a question, therefore, which is still undetermined as to whether we can regard even hemianæsthesia as indicative of hysteria. The implication of the special senses, sometimes noted in hysterical hemianæsthesia, is by no means decisive. In a part of the cases there was a distinct diminution of all the special senses on the anæsthetic side, with dyschromatopsia; in others the difference was very slight, affecting one sense only, especially the sense of smell; in other cases still, which were clearly hysterical, the special senses were wholly unaffected, or, if affected at all, difficulty was due to some local disturbance, a refractive error, old middle-ear disease, or an occluded nostril. In certain cases, too, the colour sense was perfectly normal, and the colour fields showed no special change. In a few instances the colour fields were reversed. This study of the colour fields which, I regret to say, was undertaken in only a few cases, confirms the conclusions of Mitchell and De Schweinitz (20), who found that a reversal of the colour fields was only rarely seen. It is clear, however, that the absence of disturbance of the special senses cannot exclude hysteria, in cases where there is a cutaneous hemianæsthesia, and, even, if such disturbance be noted, we must be sure that it is not due to local causes.

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If it be a question whether the disturbances of cutaneous sensibility which affect one half of the body can be regarded as an absolute indication of hysteria, the doubt becomes still greater when we have to deal with the cases, sixteen in number, where the disturbance affected one limb, both legs, or much of the body. The segmental character of anæsthesia affecting one limb, in the gauntlet, sleeve or stocking type, once thought characteristic of hysteria, is now known to occur in syringomyelia and other affections of the cord. Only a few days ago I observed such an anæsthesia of the foot and lower part of the leg, in almost precisely the area covered by a sock, in a typical case of tabes, and the boundary was as well defined as is often seen in hysteria. When the anæsthesia effects the legs alone, or most markedly, and gradually grows less as it goes up the body, fading away without any definite boundary into areas of normal sensitiveness, the hysterical nature of the disturbance is certainly questionable.

On the whole, therefore, it seems probable that the majority of cases which present disturbances of the cutaneous sensibility and contracted visual fields are hysterical; but, where these symptoms exist without the corroborative proof of other hysterical stigmata, it is doubtful whether we can assume that the case must therefore be hysterical, and it is a distinct begging of the question to claim, when a patient is clearly suffering from some other disease, that hysteria must also be present because he has hemianæsthesia and contracted fields. It is undoubtedly true that many of the symptoms which were once thought to be absolute evidence against hysteria, such as ankle clonus, facial or oculomotor paralysis, immobile pupils, incontinence of urine, and even reaction of degeneration, are said to occur in hysteria (21). Nevertheless, in spite of the great advances made in our knowledge by the French, I am disposed to agree with Rumpf (22) that the concept of hysteria has been extended to cover a much wider field than is justifiable. Souques has criticised very sharply the views of Buzzard (23) regarding the simulation of hysteria by organic disease, but it is nevertheless true that there are cases of disseminated or

diffuse sclerosis where, for a considerable period, the symptoms may seem simply those of hysteria or neurasthenia, and where it is a very difficult matter to determine whether we have to do with hysteria or a slowly progressive sclerosis. The data furnished by clinical examination may be throughout the whole course of the disease quite inconclusive, and we may have only that general impression, so impossible to describe in words yet of such value in estimating the condition before us, that the case is one of progressive structural disease of the central nervous system.

Before leaving the consideration of these various symptoms presented by neurasthenic and hysteric patients, a word must be said as to the hypothesis recently advanced by Strümpell in his remarkable article that many of them, including anæsthesia, contracted fields, exaggerated knee-jerks and an excitable pulse, are due simply to suggestion. Oppenheim's (24) answer that, in a large number of the cases, many of these symptoms are not evident except upon very careful examination, agrees fully with my own experience, and I have found in many cases that the patients had no idea that such symptoms existed. It is true that by suggestion we can often excite or remove symptoms in hysterical cases. If, however, a certain number of persons are subjected to accidents of a similar character, and, as a result, they all become nervous and suggestible, and are all subjected to examinations of a similar character, why should some present anæsthesias, contractures, paralyses and contracted fields, and others present none of these symptoms? Or, if, as Mendel claims (25), the æsthesiometer and perimeter, and careful and repeated examinations suggest and develop these hysterical stigmata, why, in a well-conducted clinique where all patients are thoroughly examined, or in the private practice of men who habitually make thorough examinations, should not these symptoms be more common?

We are not yet in a position to say why one patient becomes hysteric, and another neurasthenic, as a result of an injury, but it is certainly more reasonable to seek the difference in the patient's previous condition or in factors connected with the accident itself than in the events which

follow some time later after some indications of disease have already appeared, and it is most unreasonable to ascribe these symptoms simply to the ordinary methods of examination.

The prognosis of these two affections still remains uncertain, and our judgment in any given case is liable to error. I have made some effort to collect definite statistics concerning a considerable number of cases in order to obtain accurate knowledge. It is needless to say that the task is difficult, and I can here give only my own knowledge of the subsequent history of a certain number of cases of hysteria, as the information collected with regard to the neurasthenic cases is still scanty. I can speak of twenty-eight cases of hysteria, eighteen of which were litigation cases and ten of which were non-litigation cases. Of the litigation patients one has recovered completely, five have improved considerably, but still complain and show hysterical symptoms, seven have not improved, and five have died. Of these five, one died from unknown causes, one had considerably improved but died of intercurrent pneumonia, one was steadily growing worse and died of a low form of pneumonia, and two died of exhaustion due to hysteria. Of the non-litigation patients, three have completely recovered, four have improved, two have not improved, and one has died of intercurrent pneumonia. The one litigation patient who made a complete recovery, grew somewhat worse after the case was settled, and then, after a good many months, had a bad carbuncle on the back of the neck. On recovering from this he made a complete recovery from the hysteria. It will be seen from these figures that, so far as my own investigations have gone, the remarkable cases that recover as soon as damages are paid do not appear, although we must admit the possibility of a sudden disappearance of some or all the symptoms in a genuine case of hysteria. It is well, too, to accept with reserve the statement that a patient is well because one or several trustworthy persons have seen him walking about and attending to his affairs—a truism which would hardly seem necessary to utter to any one at all familiar with nervous diseases. If a patient still presents

the stigmata of hysteria, even though he make no complaint and attend to his affairs, he cannot be considered as a healthy man.

The much more favourable outcome of the non-litigation cases is due in part to the absence of the disturbing factors attending litigation. It must be borne in mind, however, that these cases were for the most part of a milder character, due to a less severe injury, and that the patients presented themselves for treatment early in the progress of their trouble. Even under these circumstances, however, some of the cases have not improved, but, on the contrary, have shown a progressive deterioration.

The guarded nature of the prognosis which must be rendered in a given case of traumatic hysteria is therefore evident, and it is confirmed by the long duration which we note in individual cases, a number of the cases tabulated having continued for from six to twelve years, often with some improvement, but still presenting distinct symptoms and not having regained their previous health.

There seems to be no very definite relation between the severity of the symptoms in hysteria and the gravity of the prognosis. Certainly there is no connection between the intensity of the stigmata and the subsequent history of the same. One of my fatal cases and several very protracted and obstinate cases presented stigmata so slight as to be detected only after careful examination. I am therefore much more inclined to believe that the prognosis of hysteria depends, as Binswanger states (26) of neurasthenia, not so much upon the intensity of the symptoms as upon their course of development and duration. The longer, therefore, hysteria lasts the less likelihood is there of complete recovery.

I have already stated that I am as yet unable to give definite statistics as to the subsequent history of a considerable number of cases of neurasthenia. Some of the cases certainly progress more favourably under treatment than do the cases of hysteria; and, on the whole, I think the prognosis more favourable. But, on the other hand, I have known of cases of long duration (eight, twelve, fifteen and even twenty years), so that the possibilities of grave results should always

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be borne in mind. Von Hösslin (27), indeed, speaks of the prognosis in traumatic cases as much graver than in cases of a non-traumatic nature, but this seems distinctly doubtful.

In the discussions held last summer in Germany, the general opinion was that the best treatment in these conditions was to keep the patient at work. It is undoubtedly true that in our enthusiasm for the rest-cure we are prone to overlook the fact that in a considerable number of neurasthenic and hysteric cases a work-cure is much more indicated. In the majority of cases it is also true that if the patient be allowed to sit or lie unoccupied and to brood upon his troubles, to make no effort, to study every symptom and to receive the suggestions and sympathy of his friends, his condition will rapidly become worse; but this is not the rest cure. In more than one case I have seen the condition aggravated by ill-advised attempts to work or by the necessity of daily labour to obtain a living. The good results in a number of non-litigation cases seen at the hospital were due directly to the prompt inauguration of treatment, the enforced isolation and the complete rest. With this, of course, should go a proper psychical treatment, constant encouragement, occupation and diversion, and a very guarded attempt to resume the normal occupations of life. It is often said that no improvement is likely to take place while litigation is pending. This is in a measure true, as the worry attendant upon litigation acts as a constant source of irritation; but I have in spite of this seen patients who were put under proper treatment make a very considerable gain in spite of the continuance of litigation. Within a very short time I saw a patient who was under very bad psychical influences and was steadily growing worse. Removal to the hospital, where he could be isolated and kept from these bad influences, produced a remarkable improvement, although the trial of his case was imminent.

The admitted evil influence of litigation, however, indicates to us a duty which we owe to the victims of accident. The claimants for damages have a bad name, and they are generally regarded by the outside public as swindlers and extortioners. The facts, however, show that in the great

majority of cases this stigma is undeserved. Very few of them are simulators, most of them are genuine sufferers, a considerable number of them never fully recover, and, in spite of the sensational reports from Chicago which have appeared in a recent number of a popular magazine, the amount which they receive in damages is, in my experience, inadequate, especially after the expenses of litigation are deducted.[1] It is, moreover, admitted by the majority of recent writers that the prognosis in these affections becomes graver the longer the disease lasts. In one case under my observation the injury was received in March, 1890; the case was tried in January, 1892, and it is still pending before the Supreme Court on appeal. Another case, when the patient was injured in September, 1892, has just come to trial. Such cases are perhaps extreme, but it rarely happens that a case which comes to trial in Boston, ends within two years from the time of injury. These delays are a direct source of harm to the patients and undoubtedly serve to turn the scale against recovery in a certain number of cases. It is, of course, hopeless to expect any reform in legal procedure, but it still remains our duty to protest against its evils, and to seek to obviate them by urging a prompt settlement of the patient's claims out of court, in every case when it can be accomplished without too great a sacrifice.

[1] In twenty cases of hysteria and ten of neurasthenia under my observation the average award was £1,100, the highest being £4,500. Of this it is probable the patient generally gets less than half.

REFERENCES.

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(2) STRÜMPELL. "Ueber die Untersuchung, Beurtheilung and Behandlung von Unfallkranken," Munich, 1896.

(3) The most important, and indeed almost the only Italian article upon the subject of which I have any knowledge is the excellent critical digest by SEPPILLI in the *Rivista Sperimentale di Freniatria*, XVII., p. 70, 1891.

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(4) GUINON. "Les agents provocateurs de l'hystérie." Paris, 1889.
(5) JANET. "État mental des hystériques." "Les stigmates mentaux." "Les accidents mentaux." Paris, 1892, 1894.
(6) BREUER AND FREUD. "Studien über Hysterie." Leipzig. 1895.
(7) DANA. *American Journal of Medical Sciences*, October, 1890. "The Traumatic Neuroses." "Hamilton's System of Legal Medicine," II., p. 340.
(8) PITRES. "Leçons cliniques sur l'hystérie et l'hypnotisme," I., p. 125.
(9) CHARCOT. "Leçons du Mardi," II., p. 138.
(10) WILBRAND UND SÄNGER. "Ueber Sehstörungen bei functionellen Nervenleiden," p. 73.
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(12) CHARCOT. *Op. cit.* II., p. 15.
(13) CHARCOT. *Op. cit.*, II., p. 31.
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(15) CHARCOT. "Clinique des maladies du système nerveux," I., p. 27.
(16) GILLES DE LA TOURETTE. "Traité clinique et thérapeutique de l'hystérie," I., p. 218.
(17) THOMSEN UND OPPENHEIM. *Archiv. fur Psychiatrie*, XV., p. 559.
(18) CHARCOT. "Leçons du Mardi," II., p. 469. "Clinique des maladies du système nerveux," I., p. 294.
(19) SOUQUES. "Contribution à l'étude des syndromes hystériques 'simulateurs' des maladies organiques de la moelle épinière," p. 7.
(20) MITCHELL AND DE SCHWEINITZ. *American Journal of the Medical Sciences*, November, 1889.
(21) PATRICK. *Art. cit.*
(22) RUMPF. *Neurologisches Centralblatt*, June 15, July 1, 1896.
(23) BUZZARD. "On the Simulation of Hysteria by Organic Disease of the Nervous System."
(24) OPPENHEIM. "Der Fall N."
(25) MENDEL. *Neurologisches Centralblatt*, October 15, 1896.
(26) BINSWANGER. "Die Pathologie und Therapie der Neurasthenie," p. 333.
(27) MÜLLER. "Handbuch der Neurasthenie," p. 197.

PROCEEDINGS OF THE SOCIETY.

At a Pathological Meeting held on October 28th, the following Communications were made :—

Drs. Sanger Brown and Adolf Meyer.—"Morbid Anatomy in a case of Hereditary Ataxy of the type described by Sanger Brown." (Communicated by Dr. J. A. Ormerod.) The following changes were described :—

(1) Excess of corpora amylacea in the posterior roots and posterior half of the cord, and in the superficial parts of the anterior columns.

(2) Increase of neuroglia in the subpial layer, and in the degenerated parts of the cord.

(3) Certain minute changes in the cells of Clarke's columns (not shown in present specimens).

(4) Degeneration (limited to the upper parts of cord) in *certain portions of the posterior columns*, viz., near mesial septum, superficial parts of Goll's columns, borders of Burdach's and Goll's columns; in the cerebellar tracts, and slightly in the antero-lateral tracts.

(5) Similar degeneration in the lower part of medulla.

(6) In some districts of cerebellar cortex, decrease in the number of Purkinje's cells.

Drs. Samuel Gee and H. H. Tooth.—"Case of Hæmorrhage in the region of the pons Varolii almost entirely on the right side."

The lesion has caused complete destruction locally of—(1) The right lemniscus, with many transverse fibres of the pons but not the pyramids. (2) The right posterior longitudinal bundle completely, and partially (? by pressure) the left. (3) Cranial nerves right v. motor, vi. and vii.

Secondary degeneration (1) Of the right lemniscus brainwards to the optic thalamus, also to a lesser degree of the left. (2) Of the posterior longitudinal bundles brainwards to the optic thalamus, showing also a crossing of the fibres in the third nucleus; posteriorly of the same bundles to become continuous with, or identical to, the anterior ground bundles of the

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lower medulla, the post-pyramidal decussation of the lemniscus being undegenerated. (3) Of the white matter of the flocculus of the cerebellum.

Dr. W. A. Turner.—"Symmetrical Softening of the Pyramids and Inter-olivary Strata." From a case of cerebral syphilis, arising probably from blocking of a branch or branches of the basilar artery. The softening extends from the lower end of the pons Varolii to the decussation of the pyramids.

Dr. F. E. Batten.—"The Effect of Marchi's fluid on Nervous Tissue which has undergone *post-mortem* change." The author showed that under the ordinary *post-mortem* conditions no changes take place which are liable to be mistaken for pathological appearances.

Dr. James Taylor and Mr. C. A. Ballance.—"Case of Tumour of the Right Lateral lobe of the Cerebellum." Forced movements to the same side, and symptoms of intense intra-cranial pressure.

Dr. Risien Russell.—(1) Tumour of the lumbro-sacral cord, with degeneration of the posterior columns on both sides, and the antero-lateral ascending tract on one side. (2) Degenerations in the spinal cord after removal of a large tumour from one cerebral hemisphere. Though degenerate fibres were present in the crossed pyramidal tract on the side of the lesion, none could be seen passing to it at the decussation of the pyramids, where all the degenerate fibres appeared to cross to the opposite lateral column. The degeneration in the direct pyramidal tract on the side of the lesion was traced into the sacral cord.